Supplement to

THE MEDICAL JOURNAL OF AUSTRALIA

No. 1. SATURDAY, AUGUST 13, 1927. SYDNEY.

Transactions of the Australasian Medical Congress

(British Medical Association)

Second Session: Dunedin: February 3 to 10, 1927

Transactions of the Australasian Medical Congress
(British Medical Association)

Second Session: Dunedin, February 3 to 10, 1927

No. 1. SATURDAY, AUGUST 13, 1927. SYDNEY.

Office Bearers.

PATRONS.

His Excellency the Right Honourable Lord Stonehaven, P.C., G.C.M.G., LL.D., Governor-General of the Commonwealth of Australia.
His Excellency General Sir Charles Fergusson, Bart., G.C.M.G., K.C.B., D.S.O., M.V.O., LL.D., Governor-General of the Dominion of New Zealand.
His Excellency Admiral Sir Dudley de Chair, K.C.B., Governor of New South Wales.
His Excellency the Right Honourable Lord Somers, K.C.M.G., D.S.O., M.C., Governor of Victoria.
His Excellency Sir G. T. M. Bridges, K.C.B., K.C.M.G., D.S.O., Governor of South Australia.
His Excellency the Honourable William Lennon, Lieutenant-Governor of Queensland.
His Excellency Sir James O'Grady, K.C.M.G., O.B.E., Governor of Tasmania.
His Excellency Sir William Robert Campion, K.C.M.G., D.S.O., Governor of Western Australia.
His Excellency Sir Eyre Hutson, K.C.M.G., Governor of Fiji.
His Excellency the Honourable J. H. P. Murray, C.M.G., Lieutenant-Governor of Papua.
John Macmillan Brown, Esq., M.A., LL.D., Chancellor of the University of New Zealand.
T. K. Sidey, Esq., B.A., LL.B., M.P., Chancellor of the University of Otago.

PRESIDENT.

L. E. Barnett, C.M.G., F.R.C.S. (Eng.).

VICE-PRESIDENTS.

Sir Joseph Verco, M.D. (Lond.), F.R.C.S. (Eng.), President of the Intercolonial Medical Congress, 1887.
Francis Antill Pockley, M.D. (Edin. and Syd.), M.R.C.S. (Eng.), President of the Australasian Medical Congress, 1911.
Arthur C. Purchas, M.B., C.M. (Edin.), M.R.C.S. (Eng.), President of the Australasian Medical Congress, 1914.
The Honourable William F. Taylor, M.D. (King.), M.R.C.S. (Eng.), D.P.H., R.C.S. (Lond.), President of the Australasian Medical Congress, 1920.
Sir George A. Syme, K.B.E., M.S. (Melb.), F.R.C.S.E., F.A.C.S., President of the Australasian Medical Congress, 1923.
W. T. Hayward, C.M.G., M.R.C.S., L.K.Q.C.P.I., LL.D., Vice-President of British Medical Association, ex-Chairman of Federal Committee.
R. H. Todd, M.D., B.S., Dip. State Med. (Dub.), F.R.C.S.I., Vice-President of British Medical Association.
H. E. Gibbs, M.D. (Edin.), F.R.C.S.E., Vice-President of British Medical Association.
Surgeon-Captain C. A. Gayer-Phipps, R.N., Director of Naval Medical Services, Commonwealth of Australia.
Major-General G. W. Barber, C.B., C.M.G., D.S.O., V.D., Director-General of Army Medical Services, Commonwealth of Australia.
J. H. L. Cumpston, M.D., Ch.B. (Melb.), D.P.H. (Lond.), Director-General of Health, Commonwealth of Australia.
Colonel R. Tracy Inglis, C.B.E., T.D., M.B., Ch.B. (Melb.), Director-General of Army Medical Services, Dominion of New Zealand.
T. H. A. Valintine, M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.P.H., Director-General of Health, Dominion of New Zealand.
R. B. Wade, M.D., Ch.M. (Sydney), representing the New South Wales Branch.
A. L. Kenny, M.B., Ch.B. (Melb.), representing the Victorian Branch.
G. P. Dixon, C.B.E., M.B., Ch.M. (Sydney), representing the the Queensland Branch.
R. C. E. Atkinson, M.B., Ch.B. (Edin.), D.P.H. (Camb.), representing the Western Australian Branch.
H. S. Newland, C.B.E., D.S.O., M.B., M.S. (Adel.), F.R.C.S., representing the South Australian Branch.
D. H. E. Lines, M.B., Ch.B. (Melb.), representing the Tasmanian Branch.
J. S. Elliott, M.D. (Edin.), representing the New Zealand Branch.

HONORARY TREASURER.

D. W. Carmalt Jones, M.A., M.D. (Oxon.), F.R.C.P., Dunedin, New Zealand.

HONORARY GENERAL SECRETARY.

W. P. Gowland, M.D., B.S. (Lond.), F.R.C.S., Dunedin, New Zealand.

HONORARY ASSOCIATE SECRETARY.

A. M. Drennan, M.D. (Edin.), F.R.C.P.E., Dunedin, New Zealand.

HONORARY LOCAL SECRETARIES.

New South Wales: F. Brown Craig, M.B., M.S. (Syd.), F.R.C.S.E., 149, Macquarie Street, Sydney.
Queensland: E. S. Meyers, M.B. (Syd.), B.M.A. Buildings, Adelaide Street, Brisbane.
South Australia: R. L. Thorold Grant, M.B., B.S. (Adel.), Kingsmead, Brougham Place, North Adelaide.
Tasmania: E. Brettingham Moore, M.B., Ch.M. (Syd.), 149, Macquarie Street, Hobart.
Victoria: F. L. Davies, M.B., B.S. (Melb.), Medical Society Hall, East Melbourne.
Western Australia: A. Syme Johnson, M.B., B.S. (Melb.), 252, St. George's Terrace, Perth.

SECTION I.—MEDICINE.

President.

Professor A. E. Mills, M.B., Ch.M. (Syd.), New South Wales.

Vice-Presidents.

L. S. Latham, M.A., M.D., B.S. (Melb.), Victoria.
D. Gifford Croll, C.B.E., M.B. (Syd.), Queensland.
C. T. Ch. de Crespigny, M.D. (Melb.), M.R.C.P. (Lond.), South Australia.
W. Horner Nelson, B.A. (Cape Town), M.B., B.S. (Melb.), Western Australia.
T. C. Butler, M.R.C.S. (Eng.), L.R.C.P. (Lond.), Tasmania.
E. W. Giesen, M.B., B.S. (Lond.), New Zealand.

Secretary.

C. S. Murray, M.B., Ch.B. (N.Z.), Dunedin, New Zealand.

SECTION II.—SURGERY.

President.

Basil Kilvington, M.D., M.S. (Melb.), Victoria.

Vice-Presidents.

Professor F. P. Sandes, M.D., Ch.M., B.Sc. (Syd.), New South Wales.
F. A. Hadley, M.B.E., F.R.C.S., Western Australia.
E. Sandford Jackson, M.B., Ch.B. (Melb.), Queensland.
A. M. Cudmore, M.B., B.S. (Adel.), F.R.C.S., South Australia.
H. W. Sweetnam, M.B. (Melb.), Tasmania.
H. T. D. Acland, C.M.G., C.B.E., F.R.C.S., New Zealand.

Secretary.

R. A. H. Fulton, M.B., Ch.B. (N.Z.), F.R.C.S. (Eng.), Dunedin, New Zealand.

SECTION III.—OBSTETRICS AND GYNÆCOLOGY.

President.

J. W. Dunbar Hooper, M.D. (Durh. and Melb.), L.R.C.S. and P. (Edin.), Victoria.

Vice-Presidents.

Professor J. C. Windeyer, M.B., Ch.M. (Syd.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), New South Wales.
W. A. Verco, M.B., B.S. (Adel.), South Australia.
R. Marshall Allan, M.C., M.D. (Edin.), F.R.C.S.E., L.M. (Dubl.), Queensland.
E. Brettingham Moore, M.B., Ch.M. (Syd.), Tasmania.
Henry Jellett, M.D., F.R.C.P.I., New Zealand.

Secretary.

C. North, M.B., Ch.B. (N.Z.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), Dunedin, New Zealand.

SECTION IV.—PATHOLOGY AND BACTERIOLOGY.

President.

A. H. Tebbutt, D.S.O., V.D., B.A., M.B. (Syd.), D.P.H. (Oxon.), New South Wales.

Vice-Presidents.

R. J. Bull, M.D., B.S. (Melb.), Victoria.
J. V. J. Duhig, M.B. (Syd.), Queensland.
J Dale, O.B.E., M.B., B.S. (Birm.), Western Australia.
A. B. Pearson, M.D., D.P.H. (Edin.), New Zealand.

Secretary.

Professor A. M. Drennan, M.D. (Edin.), F.R.C.P.E., Dunedin, New Zealand.

SECTION V.—PREVENTIVE MEDICINE AND TROPICAL HYGIENE.

President.

Harvey Sutton, O.B.E., M.D. (Melb.), D.P.H., B.Sc. (Oxon.), New South Wales.

Vice-Presidents.

T. W. Sinclair, M.D., B.S. (Melb.), D.P.H. (Lond.), Victoria.
R. W. Cilento, M.D., B.S. (Adel.), D.T.M. and H., New Guinea.
R. C. E. Atkinson, M.B., Ch.B. (Edin.), D.P.H. (Camb.), Western Australia.
M. H. Watt, M.D., D.P.H. (N.Z.), New Zealand.

Secretary.

Professor C. E. Hercus, D.S.O., O.B.E., M.D., B.D.S., D.P.H. (N.Z.), Dunedin, New Zealand.

SECTION VI.—OPHTHALMOLOGY.

President.

A. M. Morgan, M.B., B.S. (Adel.), F.A.C.S., South Australia.

Vice-Presidents.

E. C. Temple Smith, M.B., Ch.B., F.R.C.S.E., D.O. (Oxon.), New South Wales.
J. Lockhart Gibson, M.D. (Edin.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), Queensland.
D. D. Paton, M.A. (Edin.), M.B., Ch.B. (Edin.), D.O. (Oxon.), Western Australia.
D. H. E. Lines, M.B., Ch.B. (Melb.), Tasmania.
Leonard Mitchell, M.D. (Melb.), Victoria.
G. Fenwick, O.B.E., F.R.C.S., New Zealand.

Secretary.

A. J. Hall, M.B., Ch.B. (N.Z.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), Dunedin, New Zealand.

SECTION VII.—OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

President.

R. H. Pulleine, M.B., Ch.M. (Syd.), South Australia.

Vice-Presidents.

S. A. Ewing, M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.P.H. (Camb.), Victoria.
W. C. Mansfield, M.B., Ch.M. (Syd.), F.R.C.S.E., New South Wales.
W. N. Robertson, C.B.E., M.B., C.M. (Edin.), F.A.C.S., Queensland.
H. B. Gill, M.B., B.S. (Melb.), Western Australia.
G. H. Hogg, M.D. (Edin.), Tasmania.
J. Hardie Neil, D.S.O., M.B., Ch.B. (N.Z.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), F.A.C.S., New Zealand.

Secretary.

W. E. Carswell, M.B., Ch.B. (N.Z.), F.R.C.S., Dunedin, New Zealand.

SECTION VIII.—NEUROLOGY AND PSYCHIATRY.

President.

Oliver Latham, M.B. (Syd.), New South Wales.

Vice-Presidents.

W. Ernest Jones, M.R.C.S. (Eng.), L.R.C.P. (Lond.), Victoria.
D. M. McWhae, C.M.G., C.B.E., M.D. (Melb.), M.R.C.P. (Lond.), Western Australia.
Professor J. P. Lowson, M.A., M.D. (Edin.), Queensland.
P. Chisholm, L.R.C.S., L.R.C.P. (Edin.), New Zealand.

Secretary.

S. A. Moore, M.D. (N.Z.), M.R.C.P. (Lond.), Dunedin, New Zealand.

SECTION IX.—PÆDIATRICS.

President.

A. Jefferis Turner, M.D. (Lond.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.P.H. (Camb.), Queensland.

Vice-Presidents.

H. D. Stephens, M.D., M.S. (Melb.), F.A.C.S., Victoria.
R. H. Crisp, M.D., B.S. (Melb.), Western Australia.
J. M. Gill, M.D. (Lond.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), New South Wales.
Sir F. Truby King, C.M.G., M.B., C.M., B.Sc. (Edin.), New Zealand.

Secretary.

E. H. Williams, M.B., Ch.B. (N.Z.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), Dunedin, New Zealand.

SECTION X.—NAVAL AND MILITARY MEDICINE AND SURGERY.

President.

Major-General G. W. Barber, C.B., C.M.G., D.S.O., V.D., M.R.C.S. (Eng.), L.R.C.P. (Lond.), Victoria.

Vice-Presidents.

Colonel R. M. Downes, C.M.G., V.D., M.D., M.S. (Melb.), Victoria.

Lieut.-Colonel J. S. Purdy, D.S.O., V.D., M.D. (Aberd.), D.P.H. (Camb.), F.R.S.E., F.R.G.S., New South Wales.
Colonel G. W. Macartney, D.S.O., M.B., Ch.M. (Syd.), Queensland.
Colonel W. W. Giblin, C.B., V.D., M.R.C.S. (Eng.), L.R.C.P. (Lond.), Tasmania.
Colonel R. Tracy Inglis, C.B.E., T.D., M.B., Ch.B. (Melb.), New Zealand.

Secretary.

Colonel E. J. O'Neill, C.M.G., D.S.O., V.D., M.B., F.R.C.S.E., Dunedin, New Zealand.

Section XI.—Orthopædics.

President.

N. D. Royle, M.D., Ch.M. (Syd.), New South Wales.

Vice-Presidents.

W. Kent Hughes, M.B. (Lond.), Victoria.
H. S. Newland, C.B.E., D.S.O., M.B., M.S. (Adel.), F.R.C.S., South Australia.
A. V. Meehan, M.B. (Syd.), F.R.C.S.E., Queensland.
D. S. Wylie, C.M.G., C.B.E., M.B., F.R.C.S., New Zealand.

Secretary.

J. Renfrew White, M.B., Ch.M. (N.Z.), F.R.C.S., F.A.C.S., Dunedin, New Zealand.

Section XII.—Radiology and Medical Electricity.

President.

S. S. Argyle, M.B., B.S., M.R.C.S. (Eng.), L.R.C.P. (Lond.), M.L.A., Victoria.

Vice-Presidents.

A. T. Nisbet, M.B., Ch.M. (Syd.), D.P.H. (Camb.), Queensland.
D. I. R. Smith, M.B., Ch.M. (Syd.), Western Australia.
H. R. Sear, M.B. (Syd.), New South Wales.
H. C. Nott, M.B., B.S. (Adel.), D.M.R.E. (Camb.), South Australia.
F. J. Stansfield, M.R.C.S. (Eng.), L.R.C.P. (Lond.), Tasmania.
N. McDougall, M.B., Ch.B. (Glasg.), New Zealand.

Secretary.

C. C. Anderson, M.B., Ch.B. (N.Z.), D.M.R.E. (Liv.), Dunedin, New Zealand.

Committees.

The President, the General Secretary, the Associate Secretary and the Treasurer are *ex officio* members of all committees and sub-committees.

Executive Committee.

The President.
The General Secretary.
The Associate Secretary (representing also the Museum Committee).
The Treasurer.
The President of the New Zealand Branch of the British Medical Association, F. S. Batchelor.
Emily Siedeberg (representing Medical Women and the Ladies' Committee).
E. H. Williams (representing the Entertainment Committee).
R. I. Ritchie (representing the Reception Committee).
J. Malcolm (representing the Printing and Publishing Committee).
W. Newlands (representing the Trade Exhibits Committee).
R. A. H. Fulton (representing Secretaries of Sections).

Finance Committee.

The President. — The Associate Secretary.
The General Secretary. — The Treasurer (Secretary).

Museum Committee.

Gordon Bell (Secretary). — C. E. Hercus.
A. M. Drennan.

Entertainment Committee.

G. Barnett. — W. Marshall Macdonald.
J. S. Elliott (Wellington). — K. Ross.
Sir Lindo Ferguson. — E. H. Williams (Secretary).
J. A. Jenkins.

Reception Committee.

G. Fitzgerald. — R. I. Ritchie (Secretary).

Sections Committee.

C. C. Anderson. — W. Marshall Macdonald.
F. S. Batchelor. — S. A. Moore.
W. E. Carswell. — C. S. Murray.
A. M. Drennan. — C. North.
Sir Lindo Ferguson. — E. O'Neill.
F. Fitchett. — F. R. Riley.
R. H. A. Fulton. — J. R. White.
A. J. Hall. — E. H. Williams.
C. E. Hercus.

Ladies' Committee (Executive).

Lady Ferguson, Dr. Siedeberg, Mesdames Barnett, Batchelor, Fitchett, Newlands, North, Riley, Ross and O'Neill (Secretary).

General Congress Committee.

The members of the Otago Division of the New Zealand Branch of the British Medical Association.

List of Members of Congress.

Acland, H. T. D.
Adey, J. K.
Aitken, W.
Aldred, B. F.
Allan, R. F.
Allan, R. M.
Allen, I. M.
Ambrose, T.
Anderson, C. C.
Anderson, E. G.
Anderson, J. R.
Anderson, W. A.
Apperly, F. L.
Appleby, F. J.
Argyle, Stanley.
Armit, H. W.
Armstrong, J. S.
Arthur, S. H.

Bage, Ch.
Baker-McLaglan, E. S.
Baldwin, G. R.
Barber, G. W.
Barnett, L. E.
Barnett, G. M. F.
Barr, V. H.
Barrett, Sir J.
Barrington, F.
Batchelor, F. S.
Bathgate, W. D.
Bathgate, W. I.
Baxter, R. H.
Beatty, H. R.
Beedie, W.
Begg, R. C.
Bell, F. G.
Bell, Geo.
Bell, M. E.
Benham, P. B.
Bevan Brown, F. V.
Biggs, A. C. B.
Bignell, F. L.
Billcliff, H. S.
Bisset, J. A.
Black, A. G.
Blackmore, G. J.
Blackwood, F. M.
Boag, N. F.
Borrie, W. H.
Bostock, J.
Bowerbank, F. T.
Bowie, J. T.

Boyd, J. R.
Brass, A. L.
Brett, P. G.
Brewis, A. S.
Brown, E. E.
Brown, S. E. V.
Brown, W. M.
Brummitt, E. A.
Bryson, Eliz.
Buchanan, G. A.
Buchanan, Lyle.
Buck, P. H.
Buckley-Turkington, E. A.
Bull, R. J.
Bull, S. A.
Burns, C. R.
Butler, T.
Byrne, Ethel.

Cameron, D. A.
Cameron, P. D.
Campbell, G. B.
Campbell-Smith, A.
Carbery, A. R. D.
Carswell, W. E.
Caselberg, A. L.
Cattell, S. R.
Champion, Ed.
Champtaloup, Mary A.
Chenery, A.
Cherry, P. T. S.
Chisholm, P.
Church, R. A.
Cleland, J. B.
Cohen, L. D.
Coldicutt, C. E. A.
Cole, J. M.
Collins, Hon. W. E.
Connor, J. I.
Cook, D.
Cooke, E. J.
Corkill, T. F.
Cotterill, A. J.
Coutts, Mary.
Cowan, D. R. W.
Cowie, J. A.
Crago, W. M.
Craig, F. B.
Craig, R. G.
Crawford, A. J.
Crawford, S. W.
Crawshaw, J. H.
Crisp, R. H.
Croll, D. G.
Cudmore, A. M.

Dansey, St. J. W.
D'Ath, E. F.
Davidson, J. K.
Davies, F. L.
de Courcy, M. H. Grace.
De Garis, Mary C.
de Latour, C. E.
Denehy, W. J.
Devine, H. B.
Dick, J. A.
Diethelm, O. A. A.
Dight, W. B.
Dixon, G. P.
Donaldson, A. R.
Donaldson, J. B.
Douglas, Alex.
Douglas, Hugh.
Drennan, A. M.
Dunstone, H. E.
Durie, Beatrix.

Easterfield, Helen.
Edgar, J. J.
Edgar, R. W.
Edye, B. T.
Elliott, J. S.
Ellis, Constance.
Elmslie, Alex.
Eudey, W. S.
Ewen, H. B.
Ewing, S. A.

Fairclough, W. A.
Falconer, A. R.
Felkin, R. W.
Fenwick, D. E.
Fenwick, G. E. O.
Fenwick, P. G.
Fergus, Thomas.
Ferguson, Sir H. L.
Fisher, G. J. S.
Fisher, Welby.
Fisher, W. B.
Fitchett, F.
Fitzgerald, Elspeth.
Fitzgerald, G. P.
Fitzgerald, J.
Fitzgerald, R. S. J.
Fletcher, W. M. A.
Foster, P. S.
Fox, A. W.
Foxton, H. V.
Frazer, J. L.
Fulton, N. E. H.
Fulton, R. A. H.
Furber, R. I. (D.S.O.).

Geerin, S. L.
Gibbs, H. E.
Gibbs, S. A.
Gibson, A. J.
Gibson, H. R.
Gibson, J. L.
Giesen, E. W.
Gill, H. B.
Gillett, G. E.
Gillies, S.
Gilmour, J. R.
Gilmour, W.
Glasgow, W. T.
Good, R. N. S.
Gordon, Doris C.
Gordon, C. H.
Gordon, K. F.
Gordon, W. P. P.
Gow, Peter.
Gower, G. Wm.
Gowland, W. P.
Grant, A. McG.
Gray, H. J.
Gray, H. P.
Green, Samuel.
Greenslade, C. M.
Greenwood, H.
Gribben, S. L. H.
Gunn, Eliz. C.
Gunn, R. M.
Gunson, E. B.
Guthrie, John.
Guthrie, R. N.
Gutteridge, E. W.

Hadley, F. A.
Halford, A. C. F.
Hall, A. J.
Halley, Ida G. M.
Hamilton, I.
Harding, H. W. L.
Harper, G. C.
Harris, S. H.
Harrison, T.
Harty, E. R.
Harty, G. W.
Haslett, S. L.
Hector, C. M.
Henry, C. D.
Hercus, C. E.
Heydon, G. A. M.
Hill, Florence M.
Hipsley, P. L.
Hogg, A. W.
Hogg, R. H.
Holmes, H. G.
Holmes, H. I.
Hooper, J. W. D.
Horne, George.
Hosking, A.
Hotop, F.
Hughes, W. K.

Inglis, W. K.
Irving, Wm.
Irwin, E.
Irwin, Mary E.
Iverach, J. A. D.
Izard, A. W.

Jackson, E. S.
Jackson, C. P.
Jameson, A. B.
Jamieson, J. P. S.
Jellett, Henry.
Jenkins, J. A.
Jermyn, F. D.
Johnson, A. S.
Johnson, T. W. J.
Johnston, A. O.
Johnston, W. D. S.
Johnston, W. P.
Jones, D. W. C.
Jones, W. E.

Kellaway, C. H.
Kenny, A. L.
Kidd, A.
Kilvington, B.
King, Sir F. T.

Lange, E. R.
Latham, L. S.
Latham, Oliver.
Lawrence, H. F.
Lawrence, W. R.
Leahy, J. P. D.
Lethbridge, H. O.
Lewis, J. B.
Lillie, C. O.
Lindon, E. H.
Lindsay, A. B.
Line, L. A.
Lipscomb, T. W.
Long, W. J.
Loughman, J. R.
Louisson, W. G.
Love, Jos.
Low, D. C.
Lynch, P. P.
Lyth, C. E. W.

Macartney, G. W.
MacCallum, P.
MacCormick, K.
MacDiarmid, J. B.
Macdonald, J. G.
Macdonald, W. J.
Macdonald, W. M.
Macfarlane, A. A.
MacGibbon, T. A.
Mackay, D.
Mackay, E. A.
Mackeddie, J. F.
Macknight, C. M.
Macky, J. C. D.
Maclean, G.
Macnamara, Jean.
Macpherson, J.
Maguire, F. A.
Malcolm, John.
Manning, L. S.
Mansfield, W. C.
Marchant, E. L.
Marks, A. H.
Marshall, A. McP.
Marshall, J. A.
Marshall, H. H.
Martin,
Matison, E. A.
Maudsley, H. F.
Meade, F. G.
Mecredy, R. J. R.
Meehan, A. V.
Mercer, W. B.
Meyers, E. S.
Mickle, A. F. J.
Mill, Thos.
Miller, A. E.
Miller, G. I.
Mills, A. E.
Minnett, R. B.
Minogue, S. J.
Mitchell, B. Aileen.
Mitchell, D. M.
Mitchell, L. J. C.
Moody, A. S.
Moore, S. A.
Moppett, W.
Morgan, A. M.
Morgan, F. G.
Morice, C. G. F.
Morkane, C. F.
Morris, J. N.
Morton, A. S.
Mullin, W. J.
Murray, C. S.
Murray, D. N. W.
Muston, W. K.
Myers, D. F.

McAra, Wm.
McArthur, A. N.
McClemens, Dorothy.
McCoy, H. A.
McDougall, N.
McGavin, D. J. (Sir).
McInnes, A. S.
McIntosh, A. M.
McKellar, T. G.
McKelvy, J. L.
McKibbin, T.
McLeod, N.
McRae, Rae.
McWhae, D. M.

Neill, J. H.
Nelson, W. H.
Newell, J. A.
Newing, S. J.
Newland, H. S.
Newlands, Wm.
North, Ch.
North, H. M.
Northcroft, H. M.
Nott, H. C.
Nyulasy, F. A.

O'Neill, E.
Orbell, R. S.

Paget, T. L.
Pairman, J. C.
Palmer, H. W.
Parkes, W. M.
Paterson, Ada G.
Paterson, Ch. A.
Paterson, D. M.
Pearson, A. B.
Pemberton, C. A.
Pezaro, M. G.
Pickerill, H. P.
Piness, G.
Platts-Mills, Daisy E.
Poate, H. R. G.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. M. DRENNAN.

FIGURE III.

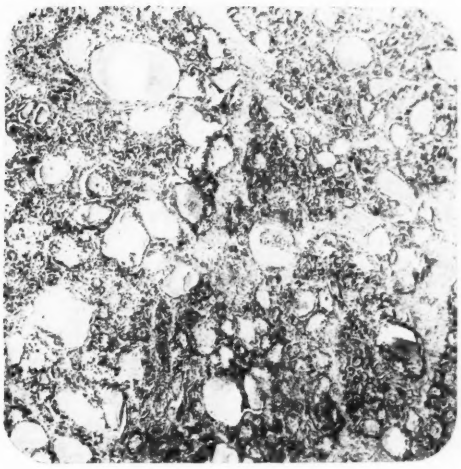


FIGURE IV.

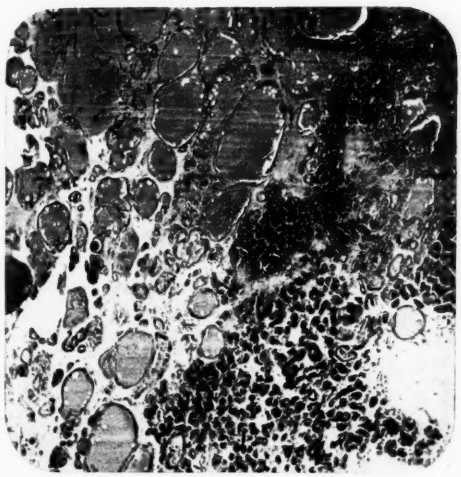


FIGURE V.

FIGURE VII.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. M. DRENNAN.

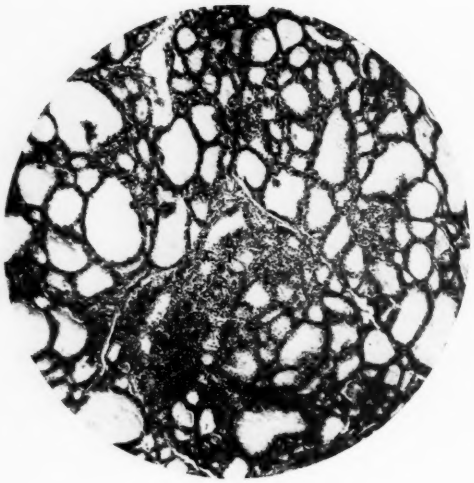


FIGURE IX.

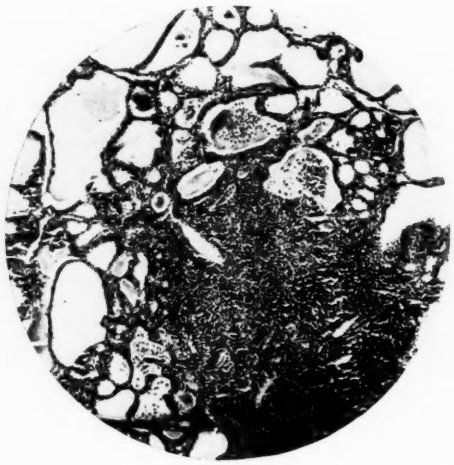


FIGURE XI.

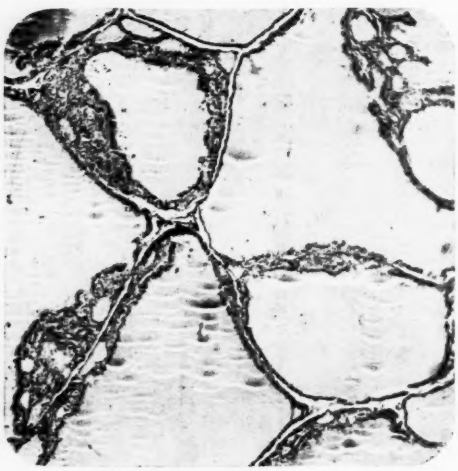


FIGURE XIII.

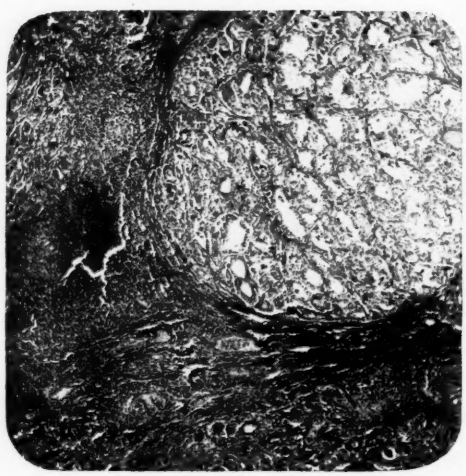


FIGURE XV.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. M. DRENNAN.

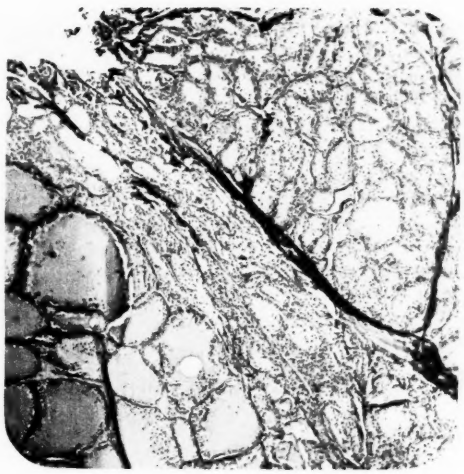


FIGURE XVI.

FIGURE XVIII.

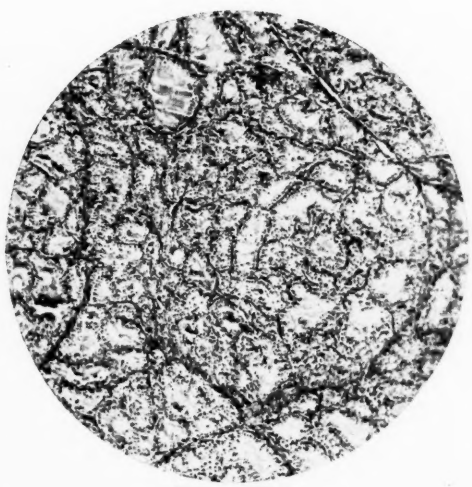


FIGURE XVII.

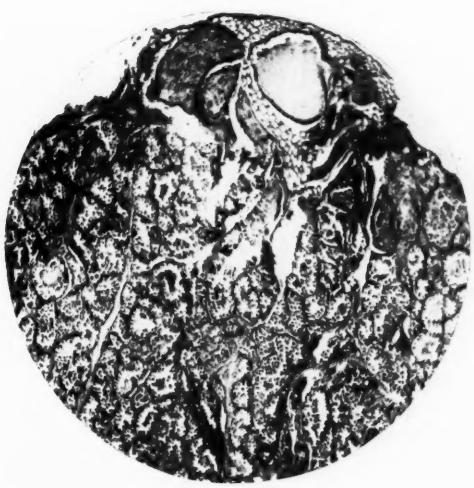


FIGURE XIX.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. M. DRENNAN.

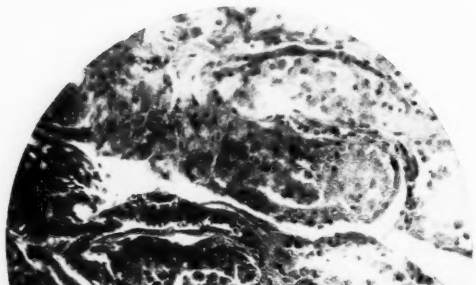


FIGURE XX.

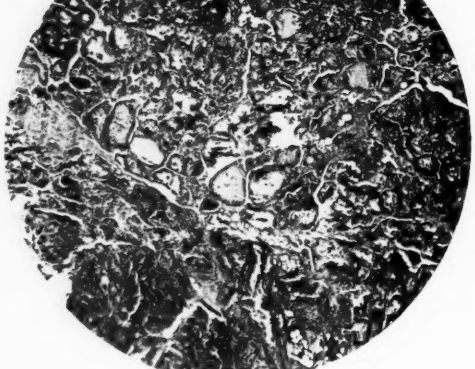


FIGURE XXI.

Pockley, A. N.
Porteous, W. J.
Pottinger, J. A.
Praagst, H. F.
Prior, N. H.
Pulleine, R.
Pullen, E. D.
Purdy, J. S.
Putnam, P. T.

Radcliffe, D. G.
Ramsay, J.
Rees, W. L.
Reid, A. A.
Reid, Ch. E.
Reid, James.
Reid, J. H.
Reid, O. J.
Rhind, S. D.
Rich, W. G.
Riley, F. R.
Ritchie, R. I.
Ritchie, T. R.
Robertson, E.
Robertson, C. H.
Robertson, H. G.
Robertson, W. N.
Rogers, J. E.
Rogers, L. H.
Ross, A. M.
Ross, Kenneth.
Rowley, E. O.
Rowley, Chas.
Rowley, Gladys.
Roy, A. B.
Royle, N. D.
Russell, R. H.
Ryan, T. F.

Sandes, F. P.
Sandston, A. C.
Saw, A. J. H.
Scannell, F. A.
Schlink, H. H.
Schwartz, Z.
Scott, E. W. K.
Scott, F. L.
Scott, J. M.
Scott, W. G.
Scoular, S.
Scrymgeour, D. N. McC.
Sear, H. R.
Sewell, S. V.
Shackleton, R. G.
Shaw, J. P.
Sheahan, J. G.
Shore, R.
Shorney, H. F.
Siedeberg, Emily H.
Simmons, W. T.
Simpson, C. J.
Sinclair, D. L.
Sinclair, R. G. B.
Sinclair, T. W.
Skerman, S.
Skirving, R. S.
Smalpage, E. S. H.
Smith, C. N.
Smith, D. R.
Smith, E. C. T.
Smith, G. W.
Smith, H. H.
Smith, S. A.
Somerville, J. E. W.
Spedding, I. N.
Speira, N. L.
Spencer, F. M.
Stanton, A. M.
Stawell, R. R.
Steenson, K. R.
Stenhouse, J. S.
Stephen, E. H. M.
Stephens, H. D.
Stevenson, Grace.
Stewart, J. G.
Stewart, R. S'C.
Stokes, A. F.
Stokes, H. L.
Stout, T. D. M.
Stout, Robert.
Stowe, W. R.
Strain, S. B. W.
Stronach, R. D.
Stubbs, E. S.
Stump, C. W.
Summons, W.
Sutcliffe, E. W.
Sutton, H.
Sweet, G. B.
Syme, Sir G. A.

Tait, J. T.
Talbot, A. G.
Talbot, G. G.
Talbot, L. S.
Taylor, Marion A. R.
Taylor, W. F.
Tebbutt, A. H.
Thomas, J. R.
Thomson, E. F.
Thompson, S. J.
Todd, R. H.
Todd, Wm.
Tracy-Inglis, R.
Trotter, A. M.
Trumble, H. C.
Tweed, M. B. M.
Turbott, H. B.
Turner, A. J.

Ulrich, F. F. A.
Unwin, W. H.
Usher, T. N.
Ussher, G. H.

Verco, W. A.

Wade, R. B.
Wade, W. R.
Wales, H.
Walker, G.
Washbourn, H. O.
Watt, M. H.
Wells, A. Q.
Wells, J. R.
Wettenhall, R. R.
Whetter, J. P.
White, E. R.
White, J. R.
Whyte, Marion.
Wiley, C. J.
Will, J. L. A.
Will, T. A.
Will, W. J.
William, W. E.
Williams, E. H.
Wilson, Alex.
Wilson, A. M.
Wilson, Mary P.
Wilson, H. M.
Wilson, T. A.
Windeyer, J. C.
Wi-Repa, T.
Wishart, W. B.
Wood, A. J.
Woodhouse, Irene.
Worrall, R.
Wylie, D. S.
Wylie, R. McA.

Young, Wm.

Zwar, B. T.

Inaugural Meeting.

The second session of the Australasian Medical Congress (British Medical Association) was formally opened in the Early Settlers' Hall, Lower Stuart Street. Sir George Syme was in the chair.

The Honourable W. Downie Stewart (Acting Prime Minister) said that in welcoming on behalf of the Government those who were attending the Congress in Dunedin he desired to express his regret that Mr. Coates, their Prime Minister, had not been able to return to New Zealand in time to open the Congress. They might have noticed, however, that since Mr. Coates had finished his attendances at the Imperial Conference he had been receiving on his own behalf doctors' degrees in the universities of the old country. He hoped that when Mr. Coates arrived back in New Zealand he would be able to prescribe for and diagnose some of the political problems he had waiting for him. He had been pleased to hear from Dr. Barnett that they had quite a number of overseas visitors with them that night who had been at a congress in Dunedin over thirty years previously. This led him to believe that the class of practitioners who not only laid down rules of health for their patients, but followed them themselves, was larger than he had supposed.

Dr. Barnett had been present at the Congress thirty years before and he thought that looking at him there was no reason why he should not be present at another congress in Dunedin held in another thirty years' time. He considered it a fortunate circumstance that the Congress was being held in Dunedin. They were a small and isolated community and the more they came into contact with the representatives from Australia, Great Britain and America, the more they would benefit. They were pleased to welcome their visitors, not only for their own sake, but because they were anxious to get more knowledge of their countries and their people.

He was glad to know that they had appointed their old friend, Dr. Barnett, as their President, because he was the best type of medical practitioner in New Zealand. From his own observation he could say that both in Australia and New Zealand the medical profession was not only of a very high standard, but that the members of the profession also made excellent citizens; they interested themselves in public affairs and particularly as patrons and supporters of the fine arts, music, painting and literature. In the latter respect Dr. Barnett was also of the best type of New Zealand practitioner.

Mr. Stewart said that the practitioners of New Zealand had not only their virtues, but their faults and one of their faults was that they fell an easy prey to company promoters and others. So far as he could see they were better at saving lives than saving money.

He had noticed a good deal of discussion in the papers as to the justification for these congresses and whether they were superfluous or not. He supposed that if any congress justified itself, it was a medical congress, because, speaking as a layman, it was in the world of medicine and surgery that great

advances were being made, with more movement of thought and progress than in any other profession. As a consequence cooperation and consultation became increasingly necessary and the more the public health was improved as a result of the advances in medical science, the more quickly would the community reach the ideal state that they all looked for and hoped for. He supposed that there was no country in which a medical congress could be held under such happy and hopeful circumstances as New Zealand. New Zealand enjoyed in the first place a climate which was extraordinarily beautiful and bracing, and good health was a national ideal and not mere Utopian dream, an ideal which was capable of approximate realization. As they were aware, their towns, consequent on the geography of the country, were of moderate size and they did not develope the dangers usually associated with large cities, such as vice, crime and squalor. He referred to the fact that the pioneers of New Zealand were the very best Anglo-Saxon stock, capable and reliant and they had striven to maintain the high standard of life of these pioneers and spoke of the happy relations of the white New Zealanders with the Maori race. The Maori race was the only native race which had not declined as a result of contact with Europeans. The Maoris, indeed, were increasing in numbers and prospering. He hoped they would have a prosperous and successful session and that every facility would be given in the community and throughout New Zealand for their visitors to form an accurate and pleasing impression of the country.

The Honourable J. A. Young (Minister of Health) said that it was a great compliment to New Zealand that the great Congress was so largely attended, not only by delegates from New Zealand and Australia, but by representatives from other parts of the world, including the United States. As Minister of Public Health he desired to join with their very able Acting Prime Minister in extending a very hearty welcome to all the visitors. A gathering of that sort was conducive to the utmost good, not only for the medical profession, but for the whole community throughout the world, because, after all, those attending the Congress were in pursuit of scientific knowledge which would be made available for the benefit of the people. Only recently he had had the honour of extending, on behalf of the Government, a hearty welcome at Auckland to Mr. S. M. Bruce, Prime Minister of Australia. He had mentioned to Mr. Bruce that the Congress was to be held in Dunedin and had asked him if he would send a message to it. He had received a radio message from Mr. Bruce who had asked that it should be read: "On behalf of the Government and the people of the Commonwealth, I desire to extend to the Australasian Medical Congress best wishes for success. Thanks to the unremitting efforts of the public-spirited members of their great profession, our citizens are gradually realizing the part which health plays in every aspect of our national life and the enormous economic waste occasioned by the inefficiency of the partly unfit. I hope and indeed feel sure that the labours of the Congress will advance us still further towards the accomplishable ideal of a happy, prosperous and healthy community." The progress, peace and happiness of the people were built up on sound physical health. The Government recognized that and every right-minded citizen with a proper sense of duty to the community recognized it also. The best assurance of health was the prevention of disease. The private practitioner was interested in promoting the health of the community, although he would not prosper very much if the community reached such a healthy state that it did not require the services of a doctor. He knew that the medical profession was cooperating with the civic authorities in endeavouring to bring about such a state of affairs and so add to the happiness of the people. The specific function of the State was to enable things to be accomplished which would insure the health of the people, in other words, the prevention of disease. There was a great opportunity for cooperation between the medical profession and the State. The State had the great responsibility of teaching the people by legislation to pave the way, so that they might do things which would save themselves from the pitfalls of disease and do things which would not expose others to disease. The great object was to eradicate disease in the community. He joined with Mr. Stewart on behalf of the Government in extending a most hearty welcome to everyone at the Congress. He trusted that the deliberations would be a monument to science and make the Congress a monument on the highway towards a solution of the many problems ahead of the profession. The Governments of New Zealand and other countries would be only too glad to do what they could to assist the people to reach a better state of health and to build up a happier and more prosperous state of society.

Sir George Syme said that exactly forty years before the idea occurred to Dr. Fulton, of Adelaide, that it would be a very good thing to have gatherings of the medical practitioners of Australia and New Zealand in congress. That idea had been warmly supported and it had materialized in the first session of what was then called the Intercolonial Medical Congress, under the presidency of Dr. (now Sir) Joseph Verco. Since then a series of congresses had been held. Sir Joseph Verco, an eminent physician, had been followed by an equally eminent surgeon, Dr. Thomas Fitzgerald. Since then there had been others, including university professors and he wished to congratulate Dr. Barnett on having been nominated by the New Zealand Branch and appointed by the Executive Committee to the position of President. He added that Dr. Barnett's reputation was world wide and it needed no words from him to add any eulogy to his work. Some who were present from overseas, had been present on the occasion of the meeting of congress in Dunedin thirty years previously. Those who had been present then, had a most extraordinary appreciation of the warmth of the welcome and the abundance of the hospitality of the Dunedin citizens. That remained as a record among the congresses that had been held since. He was confident that the Congress would exceed the success

of the previous one in Dunedin. He then vacated the chair in favour of Dr. Barnett.

Dr. L. E. Barnett then read his address as President of the Australasian Medical Congress (British Medical Association).

President's Address.

By L. E. BARNETT, C.M.G., M.B., Ch.M., F.R.C.S.,
President of the Australasian Medical Congress (British Medical Association).

I AM deeply sensible of the honour that has been conferred upon me in electing me President of this great Congress, which is representative of the medical profession of the whole of Australia, New Zealand and the outlying islands, the British Empire in the Pacific Ocean, and I thank you, Sir George Syme, for your kind and encouraging and flattering words in passing on to me the office which you have so conspicuously adorned. I shall find it difficult to live up to the high standard of presidential duty and performance that you have set me.

I welcome you all to Dunedin which is to be the scene of your labours and of some of your recreations. Those of us who are Dunedin folk, are proud of our home town, with its hills and bush, its gardens and seas. We think it has the makings of an attractive residential and university city and we hope you will enjoy your stay amongst us.

I wish to express on behalf of the Congress our gratification at seeing here the Honourable Mr. W. Downie Stewart, Acting Prime Minister for the Dominion of New Zealand, and the Honourable Mr. J. A. Young, Minister of Public Health.

It gives us great pleasure also to have with us the accredited representative of the British Medical Association, Dr. Cooper Pattin, of Norwich, whose distinction in the field of public health renders his visit to a congress of this kind particularly appropriate.

We are pleased and honoured also at having with us Dr. George Piness, of Los Angeles, the medical representative of the United States of America, a country whose magnificent scientific achievements and generous hospitality are known to most of us.

Very special welcome is due to those of our Australian visitors who were with us in Dunedin in February, 1896, exactly thirty-one years ago, at the very first Australasian Medical Congress ever held in Dunedin, and who have again crossed the Tasman Sea to take part in our proceedings.

I refer to Sir George Syme and Dr. A. L. Kenny, of Melbourne, Dr. Donaldson, of Linton, and Dr. Long, of Bendigo.

The New Zealand survivors of that Congress of long ago remember the valuable help that these men rendered to our meeting. We were glad to have them with us then; we are doubly glad to have them with us now.

It is sad to reflect that more than half of the members of the 1896 Congress have passed over to the great beyond. We deplore the deaths, particularly of many of our distinguished officials, including the President, Dr. F. C. Batchelor, the Secretary, Dr. John Halliday Scott, also Sir Harry Brookes Allen and Sir Charles Ryan.

Better men it would be hard to find.

Medical Ideals and Lay Criticism.

At the time I speak of and for long after the Australasian Medical Congress was held every three years, independently of any other organization; but beginning with the medical congress held in Melbourne a little over three years ago, it has merged itself voluntarily and I think wisely in the British Medical Association. It has thus become part of a great imperialistic, scientific institution of which His Majesty, King George V., is Patron.

Hear the words of the King when in company with Queen Mary he honoured the whole medical profession and demonstrated his goodwill towards the British Medical Association by opening their new spacious and dignified home in Tavistock Square, London, on July 18, 1925:

> I am pleased to come here today accompanied by the Queen to open the new and admirably designed House of the British Medical Association. We have always taken a sincere interest in the science and practice of medicine and surgery and I am proud to have succeeded my Father, King Edward, as Patron of your Association. Since its foundation nearly a century ago by Sir Charles Hastings your Association has shown a remarkable increase both in membership and usefulness and the well-informed and constructive criticism that it brings to bear upon the evolution of your profession is of great value. The British *Medical Acts* wisely restrict admission to the Medical Register to those who have been trained in accordance with prescribed regulations and have passed the necessary qualifying examinations. At the same time, vigilance must always be exercised in order that your profession may keep abreast with the advance of science and also preserve a high standard of professional practice and ethics. The noble purpose, the character and the skill of those engaged in the art of healing are your most precious traditions and you do well jealously to watch over such attributes.

It is a comforting reflection to us of the British Medical Association to feel that the majority of the people are of the same opinion as their sovereign in regard to the maintenance of the high ideals and traditions of medical practice. Nevertheless, we have repeated and very obvious indications that there are quite a number of people who do not see eye to eye with us, and we have to face at times very bitter antagonism towards a policy that has been devised conscientiously and with infinite pains to safeguard the interests of the general public equally with those of ourselves.

There are many who mock and deride us for our subservience to what they call a ridiculous code of medical etiquette. Funny stories about doctors are even more popular than Aberdonian anecdotes and help to fill many an odd space in magazines and

newspapers. We sometimes get amusement out of them ourselves and they at any rate do us no harm. There are some who criticize us in frank and fearless fashion, but withal reasonably and often usefully. Hostile criticism, if it is reasonable, has a more purifying and stimulating effect than friendly adulation. "A friendly eye does never see such faults" and the medical profession makes no claim to be faultless. Far from it; we are made of common clay just like other folk. We sometimes stand in need of correction. Our little medical parliaments are as liable as the greater parliaments of the people to make blunders and that is certainly a fair admission of human limitations, of human error, of human frailty. Criticism by an opposition party is a good thing in the people's parliament, just as it is a good thing in the parliament that deals with the doings of doctors. We must give consideration to the honest and reasonable criticism of the opponents of our policy, whether that criticism comes from lay or medical sources.

But we cannot help feeling resentment at the publication of gross and unwarranted statements that heap indignities upon us, that accuse us of rank dishonesty, allegations, for instance, that we are a great trades union whose chief aim is to keep the wages of its doctor members at the highest possible level, that we strangle competition by keeping out of this union the numerous and wonderful unorthodox healers who have not conformed to our requirements of a thorough medical education, that we advocate vaccinations, operations, inoculations, animal experiments and so on, not for the sake of suffering humanity, but for the benefit of our sordid selves, that we give gratuitous service to the hospitals and sick poor only because it gives us prestige and a free advertisement, that is to say, we climb over the backs of the poor into the pockets of the rich.

I have the impression that these intensely bitter and vociferous critics of ours are not only mentally perversely active, but also uncommonly healthy in body and that consequently they have not had their crude conclusions confounded by any dangerous and painful malady, accompanied by the soothing ministrations of a capable doctor. I feel certain, for instance, that if Mr. George Bernard Shaw who, though undoubtedly a dramatic genius, is always vituperative where doctors are concerned, had at any time suffered from a perforated duodenal ulcer or a gangrenous appendicitis or a pleurisy with effusion, he would not have written so mordantly and cynically about medical practice.

We judge it wise to refrain from responding in the lay press to calumniations of this kind. The piquant controversies that otherwise would arise could produce nothing but confusion. We are quite prepared to be judged by the majority. Let our lives and our actions suffice to discredit our detractors.

It has been said that human destiny is largely in the hands of the medical profession. Whether this be so or not, we must not fail to realize the sacred duty that devolves upon us, namely, the medical care of the most wonderfully beautiful and intricate living thing on earth, the human body and with it the human mind and even the human soul. We know that Nature, if unhindered by harmful customs imposed by a too luxurious civilization, can cure most illnesses and heal most wounds in human beings, just as is the rule in lower animals. We know and we preach that if people lived a more natural life from the prenatal stage onwards through babyhood to adult life, if they took more advantage of those priceless tonics, sunshine and clean, dust-free air, if feeding was more rational as regards quality, quantity, temperature and periodicity, if the drinking of alcohol was not abused, if teeth were looked after, and food well masticated and if they earned their meals by a fair measure of exercise, there would then be much less necessity to resort to the ministrations of the physician and the surgeon, to the nostrums and cure-alls of the proprietary drug vendors or to the hocus pocus of quackery.

But we doctors know that there remains a considerable and serious proportion of cases of illness, virulent microbic infections, tumours and cancers, grievous injuries and mechanical disabilities with which Nature unaided cannot cope, troubles which may at first seem slight and unimportant, but which have tragic possibilities, troubles which may, on the other hand, be overwhelming from the very outset. Not individuals alone, but whole communities suffer disaster if troubles of this degree of gravity are allowed to progress unchecked. Disease and degeneration, injuries and ill-habits and other health-destroying agencies are numerous and often complex, obscure and difficult to control. To prevent and to conquer these malign influences and to repair the ravages of illness and injury, a defending army of selected, educated and trained doctors has been organized, an army which has for its headquarters staff, its board of control, its disciplinary body, the General Medical Council and the British Medical Association.

These assemblies have always included medical representatives of eminence and special fitness for their duties. Quite recently in deference to public opinion a lay member has been added to the General Medical Council, namely Commander Hilton Young, a distinguished English politician and one of the heroes of the Zeebrugge naval attack. Lay representation on the Council is, I think, a wise provision, calculated to allay public distrust in the pronouncements of that important body.

We have, in the course of long years, evolved a scheme of medical education and training, of efficiency examinations, of ethical regulations, of safeguards against injustice, of post-graduate study and research, of preventive measures for the lessening of disease and the control of epidemics. We benefit in many ways by such a scheme, but the public benefit still more. We have the public welfare at heart just as much as, if not more than our own and that is a point which deserves reiteration.

Moreover, we do not try to interfere with public liberty. A sick man or a man who thinks he is sick, is not bound to come to one of us for treatment. He can choose a layman if he wants to; he can have the drains of his own precious body, his stomach, his bowels, his kidneys and whatnot, tinkered with by an ill-educated, self-styled healer who has not the haziest idea of the intricate mechanism of these essential organs; yet if he wants the pipes, drains or sewers of his house looked to, the law insists that he must employ a man specially trained and certified for the job—paradoxical but true.

There are a lot of peculiar people in the world who do not believe in doctors. Peculiar people have peculiar reactions and they actually do sometimes get more benefit from an unorthodox practitioner, an unqualified disciple of some weird cult or even from a rank impostor than they would from an educated and highly qualified doctor. That is a fact with a psychological basis. And so we let patients please themselves. If they do things that are too ridiculous to be tolerated, they may be censured or punished, but not by us; a lay magistrate usually attends to that.

Community service in the domain of preventive medicine, whereby the incidence of disease may be lessened and many doctors thereby rendered superfluous, has been rightly placed in the forefront of the recommendations of the General Medical Council and the British Medical Association. At this and at every British Medical Association conference public health questions will be given the greatest prominence.

We anticipate that our discussions on goître and hydatid cysts will result in a very decided lessening in the frequency of these troublesome and yet largely preventible maladies.

We shall endeavour to stimulate and correlate cancer research in the various Australasian centres. Although far more cases of cancer are cured now by operative and radiological means than in former years, chiefly because the patients are learning the wisdom of seeking treatment when the disease is in its early stage; yet, owing to the increasing prevalence of this still mysterious malady, the total death rate from cancer keeps mounting higher and higher. Yes, the cancer problem is still with us, but ever nearing solution as a result of intensive labour in a hundred fields.

I take this opportunity of congratulating the University of Sydney on the generous response of the people of New South Wales to its appeal for a Cancer Campaign Fund. With a sum of well over £100,000 a splendid plan of research can be instituted and I only hope that other scientific centres in Australasia, including Dunedin, may have the same good fortune.

And cancer is one only of many devastating diseases that call for intensive study. The governments in every civilized country now regard it as their bounden duty to encourage research that has for its object the betterment of the public health. The Royal Commission on Health for Australia, so ably presided over by Sir George Syme, have recently reported the conclusions of one of the most valuable investigations into public health matters that has ever been made and one of their recommendations and a very significant one, is that a health research council should be established and provided with an endowment of £30,000 *per annum*.

We shall at our meetings give detailed attention to the important subjects of maternal and infantile mortality, of tuberculosis and of certain tropical diseases of great public concern in northern Australia and the Pacific Islands, to questions concerning diet in health and sickness and to many other matters of special interest from the preventive medicine point of view.

I must refer also and I do it in optimistic vein, to the proposed inauguration during the currency of this Congress of the Australasian College of Surgeons, because I believe that such a college is likely to have a valuable influence on the public welfare and because I have for some years been trying to mould professional feeling in New Zealand towards something of this kind. The promoters will endeavour to combine the good points of the Royal Colleges of Surgeons of the British Isles with those of the more recently founded American College. The aims are to raise the standard of surgical efficiency, surgical endeavour and surgical ethics in both hospital and private practice and to encourage the provision of facilities in hospitals, whereby a sufficient number of suitable men can be specially trained to undertake the responsible duties of a surgeon.

In Great Britain it is quite exceptional for anyone without special training and experience to do major operations, but in the newer countries, like America, Australia and New Zealand, there is not the same restriction. In these countries major operative work is quite commonly undertaken by men who, though adequately equipped with what is popularly called nerve, lack in varying degree that knowledge and experience which are requisite in deciding such questions as: (i.) Is an operation really necessary? (ii.) When should the operation be performed? (iii.) How are the unanticipated difficulties and complications to be handled? (iv.) How should the scope of the operation be limited or extended according to circumstances?

I do not insinuate that the only competent surgeons will be those who are fellows of a college. For various reasons a man who really is a good surgeon, may not wish or may not be in a position to obtain a hall mark of this kind. The patient's choice will be unfettered. If he selects an operator who is not a fellow of a college, he may or may not get a properly trained man, but if he does choose a fellow of a college, he can rest assured that he has the services of a fully trained surgeon.

The greatest care should be exercised in the selection of the junior and senior staffs of our hospitals for many reasons and one of them has to do with the supply of trained surgeons to the community. The recognized recipe for the making of a surgeon is to take a young man who shows both

intellectual ability and manual dexterity and place him in an approved hospital, first as a house surgeon and then as assistant surgeon for several years. The passing of a high-grade academic examination is desirable, but is not essential to the performance of high-grade surgery. The main thing is good hospital work, at first under supervision and later independently.

As I said before, it is often alleged that doctors give their gratuitous services to the sick poor in hospitals for purely selfish motives, that it pays them indirectly, that the experience and prestige they gain are splendid advertisements for a lucrative practice and that consequently these honorary appointments are eagerly competed for. Now there is a measure of truth in all this, but it is not the whole truth. Painstaking, skilful and kindly hospital work does materially help in the building up of the very best class of private practice. But the kind of advertisement a hospital gives is similar to that of a shop window, exhibited, however, not to the public generally, but to those only who know the worth of the articles displayed. When a surgeon or a physician habitually shows work of good quality within a hospital, it is appraised accordingly by competent observers, that is to say by his colleagues, his students and his nurses. If, on the other hand, his work is habitually faulty, then instead of a good advertisement he gets a bad one. A hospital appointment may place a man in consultant rank, but only if he is worthy of it. The public benefit enormously by this automatic method of grading medical practitioners through the medium of their gratuitous hospital service.

Modifications in our hospital systems are now receiving serious consideration, largely stimulated by the investigations and reports of Dr. MacEachern, the American expert on hospital standardization. I hope that many of his valuable suggestions will be adopted and in particular I would like to see our public hospitals made available to all classes, but with the proviso that the sick poor shall continue to have the gratuitous services of an honorary staff in accordance with the honourable traditions of the profession. For in this as in all things we must jealously guard the great heritage of public esteem and public confidence that we owe to our predecessors.

In conclusion, let me quote what was said about us by the Right Honourable Neville Chamberlain, the Minister of Health, speaking at the London Hospital a few months ago:

> No one could doubt the fundamental seriousness of mind of any practising surgeon or physician. I do not mean that doctors have no sense of humour; God forbid! Their worst enemies have not accused them of that, but inevitably there must be in them a serious vein. They must feel that their calling is something beyond a mere means of earning one's livelihood, that they have a purpose in life which is neither selfish nor superficial and that they concern themselves not only with the cure of the individual but with the much wider task of trying to improve the general health of the people.

Ladies and gentlemen, that is the kind of criticism that is sweet to our ears; let us show that we of today are worthy of it.

Popular Lecture.

THE COMING OF THE MAORI.

By P. H. BUCK (TE RANGI HIROA), D.S.O., M.D., Ch.B.,
Director of Maori Hygiene, New Zealand.

THE coming of the Maori to New Zealand is merely an incident in the migration of the Polynesian race into the islands of the Pacific. The migrations of the Polynesians are again incidents in the dispersal of mankind. These migrations, however, are so full of romance and high endeavour that they can only be fully appreciated when compared with the migrations of other races in a primitive culture stage.

Accepting the birth place of man as being located in central Asia, we find that the very earliest types of the human race were pushed out from the common homeland in a geological period when the land surfaces were much different to what they are now. Thus palæolithic man appeared in France and Britain when there were land bridges across the Mediterranean Sea and the English Channel was a fertile valley. Whether they passed along the south or the north of Africa, they could walk to their destination. The primitive bushmen of South Africa and the negroids who followed, had no ocean tracts to negotiate. The early Tasmanian and the Australian aboriginal travelled on dry land. The first Americans travelled across a land bridge where the Behring Straits now separates two continents. Hence the earliest migrations were accomplished on foot and the large land masses of the world were first peopled by pedestrians.

The last parts to be inhabited by man were the remoter islands set in the midst of the vast Pacific, that now go by the name of Polynesia. The reason is not far to seek. Man could not walk to them and a form of transport, other than the lower extremities, had to be invented, tested and improved. A knowledge of the stars, seasons, winds and currents had to be developed empirically. Above all, daring, endurance and the spirit of high adventure had to be sown, take root and flourish in the hearts of a stone age people.

Thus the sunlit isles of the central and the eastern Pacific awaited the coming of such a race. Such a race appeared in a brown-skinned people of Caucasian descent who are now known as the Polynesians. Some of the offshoots of the Caucasian division of mankind broke away from the migration route of their main body and instead of going west, came south and east. Of their earliest travels and sojournings, we can only guess. The proto-Polynesians found themselves in south-eastern Asia and the islands of Indonesia. A certain amount of intermixture took place with people of Mongoloid stock. In this region there developed the outrigger canoe as a means of ocean transport.

Polynesian traditions go back to this period and show that there was much fighting against people

in superior numbers. The Polynesians probably voyaged out into the Pacific at the beginning of the Christian era. Whether they came by a northerly route through Micronesia or more to the south by skirting the northern coast of New Guinea, requires further research. There is a tradition of one Irapanga who sailed in a north-easterly direction to the Sandwich Islands group in the fifth century. In the islands east of Indonesia people of negroid stock were encountered and traditional narrative teems with the conflicts that took place. A certain amount of admixture took place through captured women and some physical characteristics that are regarded as Melanesian, occur amongst some branches of the Polynesians.

From the seventh to the tenth centuries great voyages were made. The traditions of the various branches teem with the exploits of various ancestors who seem to have made voyages of exploration out of the sheer spirit of adventure. Thus the Rarotongans tell of Hui-te-Rangiora who sailed down to the dark seas south of Rapa in the Austral group and saw white hills bare of vegetation, bull kelp, sea monsters and an ocean covered with material like arrowroot. This occurred in the seventh century. Two centuries later, Te Ara-tanga-nuku sailed down to the same region to verify what Hui-te-Rangiora had seen. Surely these men deserve a place in the annals of fame that are devoted to Antarctic explorers. Like Coleridge's "Ancient Mariner" they could say:

> We were the first that ever burst
> Into that unknown sea.

The Hawaiians tell of voyages to Tahiti and back. On the little island of Kahoolawe is *Ke-ala-i-Kahiki,* the Way to Tahiti. From here the canoes sailed south with *Hokupaa,* the North Star, directly astern. They lost *Hokupaa* at the *Piko-o-Wakea,* the navel of space—the equator—but new constellations in *Newe* and *Humu* served as southern guides. On the return journey they took the starboard tack against the south-east trades. When they recrossed the equator and picked up the North Star, they knew they were too far to the east. Here occurs one of the most marvellous incidents in neolithic navigation. They sailed on until the North Star was judged to be as high above the horizon as it was in the homeland they were seeking. Then they sailed west, checking their course by night. In the early voyages judging the height of the star must have been by eye. Later someone invented the sacred calabash. The calabash was cut off with a level rim. Some distance below the rim four holes at right angles were pierced on the same level. To make an observation the calabash was filled with water to the level of the holes. The eye was applied to one of the holes and, when the North Star appeared on the far rim of the calabash, it was the same height as it was in Hawaii. Why? Because the calabash had been pierced and the rim pared down in Hawaii. Admiral Rodman of the United States Navy, secured a sacred calabash and had the angle measured between the artificial water horizon and the line from the observation hole to the far edge of the rim. It was an angle of 19°. Hawaii is on the nineteenth degree of latitude north. Here we have a primitive nautical instrument of mathematical accuracy. Let us remember also that the Hawaiian Islands stretch over four degrees of latitude. The largest island with the highest mountains is the island of Hawaii. It was for the best landmark that the sacred calabash was set.

Maori tradition records the voyages of various ancestors who sailed down from Tahiti to New Zealand and went back. Most famous of these is Kupe, the discoverer of New Zealand in the tenth century. How he got here, it is hard for civilized man to understand. We travel in ocean liners and press the electric bell for the comforts of civilization. When the sea is rough, between the spasms of *mal de mer,* we say such voyages in such craft were impossible. Were it not for written record, the same spirit would deny that Drake could have circumnavigated the world with the craft that he had. Kupe was no doubt lucky, but more wonderful still, he sailed back to Tahiti to tell the tale of his discovery. Of the unsuccessful voyagers, whose bones rest on the floor of the house of *Tangaroa,* the Sea God, we know nothing. Numberless canoes, manned by equally brave and intrepid adventurers, set sail and were heard of no more. Some reached their destination and founded a new colony. Others were engulfed in the bosom of the Ocean Maid. The Rarotongans enumerate the names of canoes that left for New Zealand that we in New Zealand have no record of in tradition. They were lost in the *Moana-uriuri-o-Hiro,* the purple sea of Hiro.

But to a brave people, brown or white, the failures of others never daunt the true adventurer. To the Polynesian the ocean was his friend. He grew up beside it and in it. He could swim before he could walk. From the sea he obtained an important part of his nourishment. There were no animals to hunt. There were no herds to tend. He was a fisherman first and always and an agriculturist in season. He studied the sea in its moods, the winds and seasons to adventure forth upon it and the stars of heaven to guide him over ocean spaces. He had no fear of the sea. When a canoe was lost, he blamed the navigator, not the sea. The unsuccessful man had omitted some point in ritual or misread the signs and omens. He was justly punished. If he misinterpreted the seasons or did not read the stars aright, he suffered for his ignorance. It was probably owing partly to this psychological attitude that the Polynesians accomplished so much. No Polynesian Columbus ever had his life endangered through the fear that his crew had of falling over the edge of a square world.

The successful voyager had confidence in himself and engendered confidence in others. Thus Ru, the discoverer of Aitutaki in the Cook group, assured his crew that all would be well because he was Ru who had been belted with the red girdle of chieftainship and had studied the things of the air and the things of the sea. During a storm when the skies had been clouded for some time, Ru was

forced to invoke the assistance of *Tangaroa*, the Sea God. He did so in the following words:

O *Tangaroa*, in the limitless space of the unknown,
Clear away the clouds by day,
Clear away the clouds by night,
That Ru may see the stars of heaven
To guide him to the land of his desire.

Surely this was a true master mariner's prayer, full of confidence and self-reliance. All he wanted was a view of the stars to guide him on his course.

From Tahiti and Raiatea men imbued with the dauntless spirit of the Polynesian navigator sailed east as far as Easter Island and colonized the various groups that lay between. They sailed north to Hawaii and south-west to New Zealand.

For New Zealand the sailing directions were those handed down by Kupe. The right time of the year to sail from Tahiti was *Tatau-uru-ora*, which roughly corresponds with November. The right night was the *Orongonui*, which is the twenty-eighth of the lunar month. The direction was to keep the bow of the canoe a little to the right of the setting sun. Canoes sailed down on a visit and went back. The crews for these long sea voyages were men trained in endurance to subsist on little water. They were picked athletes for they paddled as well as sailed. Thus we find Nuku in preparing to follow Manaia to New Zealand, saying to the old men: "Pick me the men, stout of shoulder, who can bear the strain of the deep sea paddle."

New Zealand was well known in Rarotonga and Tahiti, when the historic fleet set out in 1350. Over population and a diminishing food supply had led to quarrels and wars. The virile younger chiefs, chafing against the tyranny of Uenuku, set out to found a new home in the land of high mists that lay to the south. Keeping the bows of the canoes a little to the right of the setting sun in the season mentioned, the fleet made their landfall in the Bay of Plenty when the Christmas trees were in bloom. In the month of November the sun sets in a direction south-west by west from Tahiti. Following this direction on the map the course must strike the long stretch of the North Island that extends between the North and East Capes. Thus is tradition vindicated by science.

The best known colonizing canoes are the *Tainui, Tokomaru, Kurahaupo, Te Arawa, Mataatua, Takitimu, Horouta* and *Aotea*. From the crews of these vessels have sprung the various tribes of New Zealand. Before them, however, other immigrants had landed. Much speculation exists concerning the first comers and the now practically extinct Moriori of Chatham Islands are generally regarded as part of that drift wave. The Moriori are certainly of Polynesian stock. After them came Toi and Whatonga from Tahiti. The tribes of today thus blend in their veins the blood of different migrations. It is, however, around the virile last comers that tradition proudly entwines itself. Thus the Maori poet sings:

The fame of your canoes can never be forgotten,
The canoes that crossed the ocean depths,
The purple sea, the Great Ocean of Kiwa
That lay stretched before them.

Each tribe recites the story of achievement of its own canoe. Historical chants have been handed down that were recited on the voyage or after landing. That the men stout of shoulder took pride in wielding the deep sea paddle is borne out by the following extract from the sea-chant of the Aotea canoe.

Ah, the outward lift and the dashing,
The quick thrust in and the backward sweep,
The swishing, the swirling eddies,
The foaming white wake
And the spray which flies from my paddle.

We have brought the Maori to New Zealand. In a new land with a colder climate, he grew into a virile people. He developed a characteristic art that seems peculiar to himself. In peace and war he displayed an initiative and genius that lifted him to a high position amongst peoples with a stone age culture. The stone age culture has passed away and the Maori has again to brave dangers that threaten his existence. These are the dangers of civilization in the introduction of a new culture that has changed his social and physical environment. As with the ancient voyagers, many canoes have gone down. Let us hope that by the exercise of the same courage, endurance and initiative that marked his fathers of old, the surviving canoes will weather the storm of transition and make safe landing in the haven of peace and prosperity that the new culture promises.

The Sections.

FRIDAY MORNING, FEBRUARY 4, 1927.

COMBINED MEETING.—SECTIONS I, II, IV, V AND XII.

THE INCIDENCE, ÆTIOLOGY AND PREVENTION OF GOITRE IN NEW ZEALAND.

By C. E. HERCUS, D.S.O., M.D., D.P.H.,
Professor of Bacteriology and Public Health, University of Otago.

AMONG the many advantages of such a congress as this is the opportunity it affords of indulging in a little healthy rivalry as to the relative healthfulness of our respective countries. As New Zealanders we are perhaps unduly conscious of the fact that our citizens enjoy the longest expectation of life in the world, if this can be considered a credit entry, that our babies are more tenacious of life than those of any other country and that our death rate from tuberculosis would be the lowest in the world but for the white population of South Africa. There is a debit side to our ledger, however, which we cannot conceal and therefore frankly admit. Entries here include the practically universal incidence of dental caries, the maternal mortality rate which appears to be unduly high, the undue and increasing amount of thyreoid derangement throughout certain

districts of the country. It is with the latter failure of preventive medicine that this paper deals.

Goître is no new problem in New Zealand. Elsdon Best[1] draws attention to the fact that it was recognized and named by the Maoris before the advent of the Europeans. Gilbert Mair[2] in a discussion at the New Zealand Institute refers to the presence of "Derbyshire neck" among the Maoris inhabiting the mountainous regions of the Urewera in the North Island. The disease was much less widespread, however, than it is today among the European population. The present study deals firstly with the incidence of simple and toxic goître in the European population.

Before the exact incidence of goître can be ascertained it is necessary to have some standardized method of classification and to examine the necks of all the inhabitants of a given area. In the interests of uniformity it is also desirable to have as few observers as possible. These conditions are difficult of attainment and except under exceptional circumstances goître incidence is usually determined by examining some readily accessible section of the community such as the school population. This cross-section of the population is particularly valuable, for it reduces the disturbing element of movement of population between areas to a minimum and thus reflects local conditions with as much accuracy as is possible. Such a survey was commenced in New Zealand in 1920, when Dr. Eleanor Baker and the writer examined over 12,000 school children in Canterbury and Westland and classified the condition of their

Table I.—Summary of Deaths from Diseases of Thyreoid in New Zealand, 1916-1925.

Males.

	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	Totals
Exophthalmic Goître	2	4	7	3	7	7	4	9	7	9	59
Goître (simple or undefined)	—	—	1	2	1	3	1	2	1	1	12
Myxœdema	1	—	1	—	1	1	1	—	1	—	6
Cretinism	2	1	3	—	2	—	—	—	—	—	8
Thyreoid Tumour	—	—	—	—	—	—	—	—	1	—	1
Thyreoid Insufficiency	—	—	—	—	—	—	—	—	—	2	2
Total (Males)	5	5	12	5	11	11	6	11	10	12	88

Females.

	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	Totals
Exophthalmic Goître	25	33	36	22	37	35	28	29	45	54	344
Goître (simple or undefined)	6	10	3	3	8	5	17	7	3	2	64
Myxœdema	1	1	1	—	1	1	—	1	—	2	8
Cretinism	3	3	1	2	2	1	1	2	1	—	16
Thyreoid Tumour	—	—	—	—	—	—	—	2	—	2	4
Thyreoiditis	—	—	—	—	—	—	—	—	—	2	2
Total (Females)	35	47	41	27	48	42	46	41	49	62	438
Grand Totals	40	52	53	32	59	53	52	52	59	74	526
Exophthalmic Goître (both sexes)	27	37	43	25	44	42	32	38	52	63	403
Rate per 10,000 of mean population	0·36	0·47	0·48	0·28	0·49	0·43	0·42	0·41	0·45	0·56	—

Table II.—Summary of Thyreoid Diseases Treated in Public Hospitals of New Zealand, 1916-1925.

Males.

	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	Totals
Exophthalmic Goître									15	17	—
Hyperthyreoidism	11	12	19	12	14	22	17	22	18	16	—
Toxic Goître									13	12	220
Goître (simple or undefined)	8	15	14	24	22	16	24	23	10	26	182
Other Diseases of the Thyreoid Gland	10	11	15	7	8	16	25	19	12	10	133
Total (Males)	29	38	48	43	44	54	66	64	68	81	535

Females.

	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	Totals
Exophthalmic Goître									89	128	—
Hyperthyreoidism	39	59	89	75	84	80	65	71	28	34	—
Toxic Goître									46	108	993
Goître (simple or undefined)	78	85	124	126	119	120	116	119	104	135	1,126
Other Diseases of the Thyreoid Gland	34	34	46	28	30	42	65	94	37	17	427
Total (Females)	151	178	259	229	233	242	246	284	304	422	2,546
Grand Total	180	216	307	272	277	296	312	348	372	503	3,081
Rate per thousand of total admissions	48	56	66	64	60	66	66	69	68	88	—

thyreoids according to an arbitrary scheme of classification adopted in Europe and America. These findings have been published elsewhere.[3] They served to show that there was a disquieting amount of simple goître even amongst children arriving at school at the age of five years (over 30% had well defined goîtres), that the incidence in boys and girls was equal up to the age of puberty and considerable in amount, that at puberty the female incidence rose rapidly and that the hereditary factor was an important one. This survey was extended throughout the Dominion and over 90,000 school children were examined. The results which have been published,[4] show clearly the great variation in district incidence. Goître is found to be most abundant on the more or less gravelly plains and river flats and along the narrower flood plains of our large rivers.

There is other important evidence, however, of the extent to which a district is affected by goître. The number of people dying from thyreoid disease gives valuable information, as do the admissions to public hospitals for treatment for thyreoid disease.

Dr. Watt, Deputy Director-General of Health, has produced data on these matters covering the ten years' period 1916-1925 (see Tables I and II).

It will be seen that there have been 403 deaths from toxic goîtres, 344 being in females, and that there has been an undue increase in the deaths from this cause during the last two years. There have been 3,081 cases of thyreoid disease treated in the public hospitals with a considerable increase in 1925, due principally to the increase in simple and toxic goître. Of the total admissions 1,213 or 39·3% were due to toxic goîtres and 1,308 or 42·4% to simple goître.

The incidence of hyperthyreoidism according to district is indicated in Table III. which shows the number of hospital admissions for this condition for the period 1916 to 1925. A definite relation is indicated between the incidence of toxic and simple goître. A similar parallelism has been previously noted in both Europe and America. It is worthy of note that during this period there have been twenty-four deaths from cretinism and fourteen from myxœdema.

Further evidence as to district incidence is supplied from the results of the medical examination of all males between the ages of twenty and forty-five during the period of conscription during the late war.

W. M. Ash[5] from a study of the publication mentioned above, challenges the contention that in certain districts in New Zealand there exists a goître problem of serious magnitude and considers that our endemicity is of an extremely low order. He commits the natural error of a critic with no local knowledge surveying these islands from a distance of 13,000 miles. He considers the country as a whole and ignores the extraordinary district variation. The same critic regards the majority of our thyreoid enlargements in children as physiological, although why thyreoid enlargement should be physiological in children in some districts and not in others is not explained. Physiological enlargement of the thyreoid occurs at pregnancy and during menstruation, but does not occur in children of both sexes before the advent of puberty. Furthermore, physiological enlargement does not persist after the removal of the increased stimulus to thyreoid activity. The enlargements which we record, are chronic, persistent enlargements which, as the obser-

TABLE III.—HYPERTHYREOIDISM IN NEW ZEALAND.
Admissions into Hospital During the Years 1916-1925.

Provincial District.	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	Totals
Auckland	34	58	63	76	63	77	68	72	77	140	728
Hawke's Bay	13	16	26	16	27	34	33	51	36	49	301
Taranaki	3	1	11	8	9	2	7	10	7	15	73
Wellington	28	42	43	39	42	46	60	55	83	94	532
Marlborough	14	8	12	12	6	4	7	4	5	2	74
Nelson	6	7	9	10	12	12	9	7	15	16	103
Westland	2	2	1	1	5	5	4	4	10	21	55
Canterbury	45	54	79	72	77	76	82	102	94	100	781
Otago	30	21	54	35	30	30	34	33	36	55	358
Southland	5	7	9	3	6	10	8	10	9	11	78
Dominion Totals	180	216	307	272	277	296	312	348	372	503	3,083

Cases Treated per 10,000 of Mean Population.

Provincial District.	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	Average
Auckland	0·98	1·68	2·05	1·98	1·58	1·87	1·62	1·68	1·75	3·09	1·828
Hawke's Bay	2·29	2·82	4·51	2·68	4·34	5·32	5·05	7·75	5·33	7·06	4·715
Taranaki	0·51	0·17	1·86	1·31	1·41	0·31	1·05	1·50	1·02	2·14	1·128
Wellington	1·18	1·77	1·86	1·63	1·69	1·80	2·30	2·09	3·08	3·40	2·080
Marlborough	8·20	4·68	7·24	7·00	3·36	2·18	3·73	2·05	2·52	1·00	4·196
Nelson	1·38	1·62	2·08	2·23	2·57	2·50	1·84	1·42	3·04	3·22	2·190
Westland	1·28	1·28	0·77	0·76	3·59	3·49	2·73	2·72	6·72	13·98	3·731
Canterbury	2·46	2·96	4·36	3·84	3·94	3·79	4·00	4·80	4·36	4·57	3·903
Otago	2·28	1·60	4·35	2·72	2·24	2·18	2·42	2·33	2·52	3·80	2·644
Southland	0·83	1·17	1·59	0·51	0·98	1·60	1·25	1·54	1·37	1·66	1·250
Dominion Totals	1·57	1·88	2·66	2·28	2·23	2·32	2·39	2·62	2·75	3·63	—

vation of untreated cases in girls over a period of seven years reveals, increase in size and become typical goîtres in the generally accepted sense of the term. Even if we assumed that all the smaller degrees of thyreoid enlargement in children were physiological, there would still remain in such a district as Canterbury 30% of thyreoids enlarged to such an extent as to be immediately recognized by a lay observer. From the comparatively low figure of recruits rejected for goître (1,581) Ash makes the unwarranted assumption that the child incidence is not reflected in the adult population. A brief visit to the streets of our endemic areas would speedily correct this fallacy. Owing to the kindness of Dr. McKillop, Medical Superintendent of the Mental Hospital in Christchurch, the writer was permitted to examine 851 patients, mostly adults long resident in Canterbury. The findings were arresting. Of 479 females examined 58% had well defined goîtres, while 23% showed gross deformity, in some cases producing pressure symptoms. Of the 372 males, 52% were goîtrous, 20% showing huge deformity bulging. The close approximation of the male and female incidence is an important criterion of high endemicity. In order to compare these figures with those of an area of lower endemicity Dr. Gribben, of the Seacliff Mental Hospital situated near Dunedin, gave permission for an examination of his 1,153 patients. The incidence in women was found to be 15%, only seven goîtres being classified as large, whereas in men the incidence was 5%, with only two large goîtres. On investigating the place of abode of the latter patients before admission to hospital it was found that the majority came from Clutha Valley or the Southland plains which are endemic areas.

There is still other evidence forthcoming, if it should be required, from the routine *post mortem* findings at the hospitals of the larger centres, from the incidence in animals and from the rapidity with which newcomers from non-endemic areas develope goître on entering endemic areas and *vice versa* in so far as many of the smaller goîtres are concerned.

A consideration of these facts leaves no escape from the conclusion that simple and toxic goître constitutes a real and present problem in many districts in this country.

Ætiology.

It remains to consider what local evidence may be forthcoming concerning the vexed question of primary causation. By a fortunate combination of natural circumstances Nature has set a stage as if to aid such an investigation. She has spread one hundred thousand square miles of land over thirteen degrees of latitude, wholly within the temperate zone, isolated in the south-western Pacific Ocean and comprising a remarkable range of geographical circumstance. Man has peopled this stage with two races of different origin and habit of life. Side by side dwell the indigenous Maori and the European, 98% of whom are of British stock. Unfortunately the Maori has to a great extent altered his habit of life to conform with that of the European and in consequence *inter alia* there is some evidence that the incidence of goître has increased. In an examination of one hundred and sixty-one Maori children in Canterbury the author found 14% with thyreoid enlargement and in children of the Urewera country 30%. In this latter district the diet was found to be deficient in protein and fat and devoid of marine food. Members of the same tribe living in a river valley adjacent to the sea on a well balanced diet in which shell fish appeared regularly, were found to be practically free from goître. Marine food and notably shell fish bulked largely in the diet of the coastal tribes of Maoris and this fact probably explains why tribes resident on what we now recognize as endemic areas escaped the disease. It was recognized only by the Maoris in inland tribes. It is evident, therefore, that whatever may be the cause of goître, it operates independently of race. Water does not appear to be a primary factor. In certain endemic areas, notably in Canterbury and Westland, there are different sources of water supply, artesian, shallow wells, surface water and rain water, but as shown in a previous study,[6] the incidence of goître is not affected to an appreciable extent thereby. An apparent exception was found in a district where analysis showed that sea water was to some extent mixing with the fresh water and where the incidence of goître was definitely lower than in the adjacent district. If this evidence was not sufficient to show that water was of secondary importance, several epidemics of goître amongst lambs might be cited, in which the ewes had not had access to water for several months before parturition.

Calcium, that hardy annual in the annals of goître history, does not appear to be an important factor, for the only pure limestone district in the endemic area of Canterbury shows a lower incidence.

Diet.

There is experimental evidence to show that, apart altogether from the iodine factor, diets containing excessive protein or excessive fat may produce thyreoid enlargement in animals. The work of Marine and Lenhart,[7] McCarrison[8] and Mellanby[9] clearly illustrates this point. The homogeneity of our European population would suggest that the nature of the dietary, with perhaps minor modifications on account of climatic difference, would be fairly uniform throughout the country.

The following unpublished data relating to this question are supplied by Miss E. N. Todhunter, B.H.Sc., of the Home Science Department, Otago University.

From a study of 409 menus comprising 7,741 meals collected from school children in seven widely scattered districts in New Zealand (Auckland to Gore) for an average period of one week and from families averaging five to six persons: Meat is used in most cases twice a day and in many cases three times a day. Eggs are used freely. Brown bread is seldom mentioned. Bread, butter and jam is commonly given as the only evening meal. Toast and tea appears frequently as the only breakfast food. Fruit is used freely in certain districts, others hardly at all. There is a striking lack of green vegetables; 20·4% of the families never mention them and of 7,741 meals only 9% of these include a green vegetable.

Milk is mentioned by 20·4% of the families as being used as a beverage sometimes.

Data from a milk delivery round in one of the four large centres shows that for sixty-six families (including 149 children) the average milk purchased is 0·598 pints per person per day.

Speaking generally the diets are remarkably uniform.

Toxic Infective Theories.

There is no epidemiological evidence to suggest the existence of any specific infective agent as the primary cause of goître nor is there any evidence that the inhabitants of endemic areas have some specific intestinal flora, the toxic products of which either injure the gland directly or combine with the iodine in the food, thus preventing its assimilation. Concerning this latter possibility, we have carried out certain experiments on *Bacilli coli communis* and fæcal streptococci grown on agar containing a relatively large amount of iodine and find that they have failed to absorb a demonstrable amount of

TABLE IV.—GENERAL SURVEY OF IODINE CONTENT OF FOODSTUFFS.

[The figures represent microgrammes of iodine per kilogram of fresh material (per litre for liquids), *i.e.*, parts per thousand million.]

Specimen.	I_2 Content Parts per 10^9.	Specimen.	I_2 Content Parts per 10^9.
Cereal and Cereal Products—		Oysters	880
White Bread	2	Fresh Water Trout	24
White Bread (made with iodized salt)	32	Tidal Water Trout	50
Brown Bread (from Canterbury wheat low in iodine)	12	Whitebait (young of *Galaxias attenuates*)	96
Brown Bread (made with iodized salt)	55	Fish (tinned)—	
Flour (white)	3	Pilchards (mean of 2)	320
Oatmeal	30	Salmon (British Columbia sea-run)	750
Oto	61	Sardines	360
Rice (polished)	<8	Bacon (medium fat)	15
Wheat (Australian)	14	Beef (mean of 18)	13
Wheatmeal	44	Butter	12
Root Vegetables—		Cheese	31
Beetroot (without skin)	3	Eggs (mean of 38)	94
Beetroot (with skin)	15	Glaxo (dried milk)	96
Carrot (without skin, mean of 12)	10	Cow's Milk (mean of 39)	20
Carrot (with skin, mean of 2)	19	Human Milk (mean of 18)	32
Carrot (artificial manure)	2,500	Mutton	10
Kumera	3	Rabbit	6
Marrow	6	**Leafy Vegetables—**	
Mangold (unmanured)	4	Asparagus	17
Mangold (artificial manure)	5,000	Beans (Scarlet Runner)	18
Onion	11	Cabbage (unmanured, mean of 3)	17
Parsnip (without skin, stable manure)	12	Cabbage (manured)	200
Parsnip (with skin, stable manure)	53	Celery (stalks)	17
Parsnip (with skin, unmanured)	18	Leek (mean of 2)	18
Potato (without skin)	10	Lettuce (unmanured, mean of 8)	17
Potato (with skin)	22	Lettuce (manured)	130
Turnip (manured, root with skin)	16	Lucerne (autumn)	28
Turnip (manured, leaf)	92	Silver Beet (mean of 20)	27
Turnip (unmanured, without skin)	6	Spinach (autumn)	48
Turnip(unmanured, with skin)	16	Sour Thistle (Rauriki)	32
Yam	6	Average for root vegetables without skin and unmanured	7
Fruits (fresh)—		Average for root vegetables with skin and unmanured	18
Apple (whole fruit)	6	Average for leafy vegetables (unmanured)	24
Apple (without peel and core)	4·5	**Sundries—**	
Bananas	5	Casein (thrice extracted with alcohol)	72
Grapes	7	Grass (mean of 36)	58
Lemons	3	Hay (mean of 4)	124
Oranges	2	Honey	18
Pears (whole fruit)	4	Jam	12
Tomato	8	Marmalade	10
Fruits (dried)—		Marmite	<15
Apricots	22	Seaweed (Carrageen)	48,000
Figs	12	Sugar	0
Prunes	10	Water (12 samples)—	
Raisins	32	Minimum	<1
Animal Foods—		Maximum	17
Fish (fresh)—		Wine (mean of 2)	5
Groper	72		

TABLE V.—COMPARISON OF THE IODINE CONTENT OF FOODSTUFFS IN GOITROUS AND NON-GOITROUS DISTRICTS.

Specimen.	Non-Goitrous.		Goitrous.		Difference.
	Number of Specimens Analysed.	I_2 in γ per kilogram.	Number of Specimens Analysed.	I_2 in γ per kilogram.	
Carrot	7	9·5	4	7·7	1·8
Grass	17	64	13	47	17
Lettuce	1	24	8	12	12
Silver Beet (Autumn)	6	32	1	24	8
Beef	8	16	10	9·5	6·5
Eggs	18	137	20	56	81
Cow's Milk	18	25	21	15	10
Human Milk	4	43	14	28	15
				Average	18·9

iodine. On the face of it, it would appear extremely unlikely that so many children of Canterbury should be suffering from intestinal toxæmia in contrast with the immunity of the children of Auckland and Taranaki where the incidence of goître is low. There is no evidence that other foci of infection throughout the body are of primary importance. For example, the incidence of dental caries and enlargement of tonsils and adenoids is fairly uniform throughout the Dominion.

The field work may be summarized by stating that it establishes striking differences in district incidence and this in spite of movement of population and that it gives negative answers to certain current theories of causation.

In 1921 a systematic study of the hypothesis that the disease is primarily due to deficient iodine intake was commenced in this University and the iodine factor as it occurs in soil and water and food, in excretion and in the thyreoid gland was investigated. The work was divided between the departments of geology, chemistry, home science, pathology and preventive medicine and close co-operation was maintained. The Department of Health supported the research in a liberal manner. A number of findings is now in the press and the writer is indebted to Professor G. H. F. Nuttall for permission to utilize the summaries.

Iodine is distributed in such minute amounts throughout Nature that special methods of analysis are required to detect it. The unit of quantity is the microgramme, which is one-millionth of a gramme or the equivalent of a micron in the linear scale.

The cycle of iodine in Nature was first demonstrated by the work of Chatin in 1850.[10] His work has been confirmed and extended in recent years by Fellenberg.[11] It will be seen that the sea and igneous rocks constitute the principal reservoirs of iodine, although the actual iodine content of sea water is low, 0·023 gramme per litre. The quantity of iodine to be found in the soil derived from the rocks by weathering depends upon the original iodine content, the colloidal character of the soil and on the storage of iodine by plant life. There is a definite volatilization of iodine from the surface of the land and sea into the atmosphere. This atmospheric iodine is dissolved in rainwater, snow and dew and thus returns to the soil.

At the outset we examined the iodine content of some five hundred samples of soil obtained from all districts in the Dominion. These results have been published elsewhere.[4] It was found that the amount of iodine in the soil bore an almost inverse relationship to the incidence of goître. If the figures for soil iodine and goître incidence are plotted as ordinates and abscissæ respectively and a smoothed curve drawn, the intimate connexion between the factors is apparent. When the numerous disturbing factors which would tend to obscure such a relationship, are considered, there can be no doubt that a real correlation must exist. Soils derived from the basic igneous rocks contained the most iodine, while sedimentary soils, especially if sandy or gravelly, contained least. We have shown experimentally that the power of soils to retain iodine in spite of the washing action of water depends largely upon their colloidal state. Red, brownish, loamy soils which we have found to contain the most iodine, retain the iodine in virtue of the greater absorptive power of their colloids, whereas gritty soils poor in colloids have practically no retentive power.

A general survey of the iodine content of foodstuffs was then undertaken and the results are shown in Table IV. It will be seen that the foods richest in iodine are edible seaweed, sea-fish, particularly shell-fish, eggs, wholemeal cereal products, leafy vegetables and milk. Refined cereal products, root vegetables and fruits are shown to have a low iodine content. It will be noted that the skin of root vegetables which is so constantly discarded before cooking, contains much of the iodine. The iodine content of foodstuffs grown in typical

TABLE VI.—DETERMINATION OF IODINE INTAKE PER PERSON PER DAY.

Food.	Weight in Kilograms.	I_2 Intake Calculated for Typical Non-Goitrous Area.		I_2 Intake Calculated for Typical Goitrous Area.	
		I_2 in γ per Kilogram.	Total I_2 in γ.	I_2 in γ per Kilogram.	Total I_2 in γ.
Meat	102	16	1,630	9·5	970
Fresh Sea Fish	11·8	72	850	72	850
Tinned Fish	4·6	500	2,300	500	2,300
Oysters	0·18	880	1,580	880	1,380
White Carbohydrates	76	3	230	2	150
Brown Carbohydrates	77	40	3,080	20	1,550
Milk	350	25	8,750	15	5,250
Fats	47	5	230	3	140
Eggs	14·1	137	1,930	56	790
Fresh Fruit	91	5	450	3	270
Dried Fruit	12	20	240	12	140
Root Vegetables	164	9	1,480	6	980
Leafy Vegetables	44·5	27	1,200	20	890
Water	630	2	1,260	1	630
Sundries	28 (estimated)	3	80	2	60
Total iodine intake in food			25,290		16,550
Whence I_2 intake per person per day			40·1 γ		26·3 γ
Difference			13·8 γ		

goitrous and non-goitrous districts was investigated and the results are shown in Table V. It will be seen that the foodstuffs of non-goitrous areas contain more iodine to an average extent of 18·9 milligrammes.

Metabolic experiments carried out in the home science department established the iodine intake per person per day on a well balanced diet and from these data the difference in iodine intake between goitrous and non-goitrous districts was calculated and shown to be 13·8 milligrammes as can be seen from Table VI.

During the course of these studies a clear seasonal variation was noted in the iodine content of certain animal and vegetable foods. The maximum iodine content of vegetable matter is reached in the late autumn and winter when growth is at a minimum. In the case of eggs the maximum is reached in summer and in milk in the spring, although in the latter case calving and lactation are shown to be disturbing factors of importance. Cooking is shown to have little effect on the reduction of the iodine content of seaweed, fish and root vegetables, but to reduce the content of green vegetables by about two-thirds, indicating the necessity for using the water in which green vegetables are cooked.

In order to check our findings for iodine intake, studies were carried out in iodine excretion in goitrous and non-goitrous districts. Previous work by Fellenberg had indicated that the majority of the iodine is excreted in the urine and that this might be regarded as an accurate index of the total iodine excretion at a given time. We adopted this index in our investigation. Table VII shows the relationship. The iodine content of human milk in goitrous women was shown to be lower than in the non-goitrous and a relationship was shown to exist between the amount of iodine excreted in the urine and milk during lactation.

From a consideration of this body of facts we believe that the hypothesis that goitre is caused by a deficiency of iodine has been fully sustained by the present investigation.

Prevention.

Finally arises the problem of prevention. From the investigations recorded above it appears the primary factor in the causation of goitre in this country is a deficient intake of iodine. This deficiency has been shown to be in the neighbourhood of fourteen microgrammes, though emphasis must be laid on the fact that these figures were obtained from well balanced and adequate dietaries. From the study of diets already quoted it is evident that many of the actual dietaries in use throughout the country would produce a greater deficiency in iodine. In an actual experiment carried out by Dr. Storms and Miss Todhunter over a period of a week on an average diet constructed from these diets the total iodine intake was found to fall as low as ten microgrammes.

It would appear to be a simple matter to insure that the iodine ingestion in Canterbury should be raised to the same level as that in Taranaki. Like many problems of preventive medicine, however, the solution is fraught with many difficulties. Probably the greatest of these is the very real danger which attends the indiscriminate use of iodine in a community containing a large number of people with potentially toxic goitres. The reality of this danger was demonstrated in France between 1860 and 1870[12] and later in America.[13] The increase in the incidence of toxic goitres in New Zealand during the last two years is almost certainly due to an unwise use of iodine in goitre therapy and possibly in prophylaxis. The long experience of the profession in the therapeutic use of iodine, often in massive dosage, shows that the capacity of the normal thyreoid to deal with iodine is almost unlimited. The thyreoid takes up its daily requirement of iodine and the balance is promptly excreted producing no ill effects. There will be few practitioners, however, in our endemic areas who have not had personal experience in the readiness with which an adult patient with a large goitre may develope toxic symptoms under iodine therapy. When it is realized that there is an extensive sale of proprietary goitre remedies throughout the country and that these all contain iodine in varying concentrations, there can be little wonder that toxic goitre is on the increase.

The problem before us is to supply this biological deficiency in as universal and simple a manner as possible and in such a fashion as to be harmless to the adult population affected with thyreoid disease. If practicable the most obvious method would appear to be the raising of the iodine intake by the use of foods relatively rich in this element. Food from the sea, particularly edible seaweeds of which there are several varieties in New Zealand

TABLE VII.—CORRELATION OF IODINE INTAKE WITH EXCRETION IN URINE.

District.	Number of Specimens Analysed.	Mean Result in per Litre.
Taranaki (non-goitrous)	35	49
Canterbury (goitrous)	27	25

TABLE VIII.—GENERAL SURVEY OF IODINE CONTENT OF ARTIFICIAL MANURES.

Manure.	I_2 in Parts per 10^7.
Bonedust and Blood	14
Guano (Walpole Island)	264
Ephos (Egyptian Phosphate)	12
Nauru/Ocean Phosphate	11
Naura/Ocean Phosphate Super	22
Superphosphate—	
36% to 38% sol.	22
40% to 42% sol.	25
44% to 46% sol.	33
Basic Slag	0
Special Grain Manure No. 1	36
Special Grain Manure No. 2	48
Special Rape Manure	45
Special Turnip Manure	18
Seaweed	480
Ammonium Sulphate	0
Kainit	0
Potash Salts	0
Sodium Nitrate	600

waters, would supply the deficiency if used in sufficient quantity. Sea-fish, particularly shell-fish, might as in the days of the pre-European Maori, be used regularly in the diet, although the white flesh fish are not sufficiently rich in iodine to supply the amount required. Local products relatively rich in iodine such as watercress, eggs, milk, green vegetables should be used more freely. In an endemic area it is doubtful whether this measure alone would be sufficient, as the initial deficiency lies in the soil and is reflected in the food product.

The deficiency might be supplied indirectly by the use of iodine rich manures of which there are several on the market. Table VIII gives the result of a general survey of the iodine content of artificial manures in use in New Zealand. By means of experimental plots we have been able to show that the use of such iodine rich manures as Chilean nitrate and Walpole Island guano, the iodine content of vegetables may be notably increased. This method of supplying the deficiency might be used with advantage in endemic areas, but it is an expensive method and difficult to control and one dependent on an extensive educational campaign. It might, however, be given a trial and one might suggest to manure manufacturers that the more commonly used superphosphate, bone and blood and basic slag manures might be iodized to the same extent as Chilean nitrate. It would certainly prevent goître amongst stock particularly if rock salt of a reasonably and uniformly high iodine content was on the market. At present the product is too variable to be reliable. These manures would need to be used with regularity, however, as in the type of soil on which goître thrives, the iodine is quickly removed both by the plants and by water action.

In some parts of America and according to Ash in a certain area of Derbyshire the water supply of the endemic area has been iodized by the regular addition of sodium iodide. This method has serious limitations where the incidence of goître is extensive and uneven and in an artesian area such as Christchurch where so many private wells are in use it would be inapplicable.

Two methods of prophylaxis which are now in extensive use throughout the goître world, are the addition of iodine to some article of diet in universal use throughout the country and the giving of iodine in medicinal form to the school children of an affected district. The latter method first introduced by Marine and Kimball in America was tentatively adopted in two selected schools in Christchurch in 1921 pending such time as the exact deficiency of iodine was investigated. The iodine was given in the form of sodium iodide in solution and the dose, which was given once a week for ten weeks in each of the three terms of the year, varied according to age. Amounting to 0·24 gramme (four grains) a week or over 260,000 microgrammes for the older children. The results after five years have been published in the 1926 Annual Report of the New Zealand Department of Health. They show a valuable therapeutic and preventive result. In 1923 the scheme was adopted by the New Zealand School Medical Service and potassium iodide in weekly doses of one grain or 65,000 microgrammes, was supplied free of cost to school children in endemic areas. This scheme of prophylaxis is open to serious objections. When it is remembered that the total iodine content of the normal adult thyreoid gland is approximately 30,000 microgrammes, that the remainder of the body contains 1,000 microgrammes and that the daily iodine requirement of the healthy gland is approximately 40 microgrammes or 280 microgrammes a week, it would be apparent that excessive dosage has been used. We have been supplementing the diet iodine by the weekly addition of approximately twice the iodine content of the whole body or over one hundred and eighty times the normal weekly requirement of the thyreoid. Certainly there have been no ill effects recorded in the primary schools, but if used by people with large goîtres in older age groups, such dosage would lead to serious consequences, as the State of Michigan is at present realizing.

Apart from these objections the scheme of prophylaxis is altogether too limited in scope to be acceptable as a national scheme. Concentrating as it does on the school children, it is apt to suggest to the public that prophylaxis is principally a child problem, whereas we know that the deficiency is experienced by the pregnant woman, by the child *in utero* and indeed by the whole population of an endemic district.

Finally we consider the universal use of salt iodized so as to supply the biological deficiency. Salt would seem to be the natural source of this element, for we depend upon it largely for supplying our dietetic deficiencies in connexion with another member of the halogen group. Unfortunately sea salt cannot be relied upon to furnish a reliable source of iodine. In the evaporation of salt brines the mother liquor which is removed, takes with it the natural content of iodine. An analysis of the commercial salts in use in New Zealand show that they are excessively low in iodine. To supply the deficiency by means of this natural vehicle it is necessary, therefore, to restore the iodine in carefully measured amounts. Where this measure has been adopted with proper precautions which include the prohibition of the sale of all anti-goîtrous remedies, the results have been remarkable. Thus in the Swiss Canton, Appenzell am Rhein, where iodized salt has been in universal use since February, 1922, the incidence of congenital goître has dropped from 50% to nil and there have been no ill effects recorded on the potentially toxic adult population.

At the annual meeting of the New Zealand Branch of the British Medical Association held in Auckland in February, 1924, the Government was urged to introduce into the country the use of salt iodized so as to augment the daily intake by thirty-eight microgrammes of salt, that is salt containing one part of potassium iodide in 250,000 parts, assuming the average daily intake of salt for all purposes to be five to six grammes. The measure was recommended to be a compulsory one, as the amount

involved was so infinitesimal, 0·0004% and yet so important that to leave the matter to the individual wishes of the community was felt to be unsound.

The Department of Health introduced a definition of iodized salt into the *Food and Drugs Act* in June, 1924, which stipulates that iodized salt must contain one part of potassium iodide in 250,000 parts of salt and a quantity of table and cooking salt has appeared on the market conforming to these regulations.

The use of iodized salt is, however, as yet very limited and largely confined to table use. On inquiring in certain of our largest endemic areas it would appear that iodized salt sales only comprise about 5% of the total sales. To supply the existing deficiency the salt must be used for all purposes. If confined to table use, the amount of iodine would need to be increased involving greater potential risks. Considering the large and important issues involved and the harmlessness of the measure suggested, my own feeling is in favour of the compulsory iodizing of all cooking and table salt imported into the country. It would supply the deficiency in the endemic areas in a certain and physiological manner and would not increase the iodine intake in non-endemic areas beyond one hundred microgrammes which Fellenberg has shown is the normal ingestion in some parts of Europe.

Failing this compulsory addition the writer would suggest vigorous educational propaganda in all endemic areas on the need for and the correct use of iodized salt, at the same time warning the public of the dangers involved in the indiscriminate use of iodine.

In conclusion I would emphasize the infinitesimal amount of iodine which is involved in prophylaxis and indeed in treatment where large degenerate goitres are concerned. We must learn to think of iodine in terms of microgrammes. We are confronted with still another illustration of the fact first brought under the notice of our profession by Pasteur, that the infinitely little in Nature may be of profound significance to the life of man.

References.

(1) Elsdon Best: "The Maori," Volume II., 1924, page 49.
(2) Gilbert Mair: *The Transactions and Proceedings of the New Zealand Institute*, 1877, Volume IX, page 629.
(3) C. E. Hercus and E. S. Baker: "Statistical Study of the Incidence of Goitre amongst the School Children of Canterbury and the West Coast," *New Zealand Medical Journal*, 1921, page 116.
(4) C. E. Hercus, W. N. Benson and C. L. Carter: "Endemic Goitre in New Zealand and its Relations to the Soil Iodine," *Journal of Hygiene*, 1925, page 3; December 17, 1925, page 321.
(5) W. M. Ash: "Goitre," *Journal of State Medicine*, November, 1926, page 637.
(6) C. E. Hercus and E. S. Baker: "Further Considerations on Endemic Goitre," *New Zealand Medical Journal*, 1923, page 169.
(7) D. Marine and C. H. Lenhart: "Further Observations and Experiments on the So-called Thyroid Carcinoma of Brook Trout," *Journal of Experimental Medicine*," Volume XIII, 1911, page 455.
(8) R. McCarrison: "A British Medical Association Lecture on Goitre," *The British Medical Journal*, June 22, 1924, page 989.
(9) E. Mellanby and M. Mellanby: "The Experimental Production of Thyroid Hyperplasia in Dogs," *Proceedings of the Physiological Society*, Volume LV, 1921-1922, page vii (abstracted in the *Journal of Physiology*).
(10) A. Chatin: "*Sur l'Iodi de l'Atmosphère*," *Comptes Rendus de l'Académie de Science*, Volume L, 1923, page 420.
(11) T. von Fellenberg: "*Untersuchungen über den Jodgehalt der Luft*," *Biochemische Zeitschrift*, *Band* CLII, 1924, *Seite* 116.
(12) Baillayer: "*Enquête sur le Goître et le Crétinisme*," Paris, 1873, page 627.
(13) C. L. Hartrock: "Iodized Salt in the Prevention of Goitre," *The Journal of the American Medical Association*, 1926, page 1334.

THYREOID ENLARGEMENT IN AUSTRALIA: INCIDENCE, ÆTIOLOGY AND PREVENTION.

By HARVEY SUTTON, O.B.E., M.D.,
Principal Medical Officer, Department of Education, New South Wales.

THE incidence of thyreoid enlargement in Australia has not been fully worked out. We have fairly complete figures from the examination of school children in New South Wales, South Australia, Tasmania and a good deal of experience in Victoria, Western Australia and Queensland. *Questionnaires* issued in Tasmania to practitioners by Purdy and later repeated by Morris have been useful. We may safely say that simple enlargement of the thyreoid is a rarity in South Australia (Halley, Cleland, Ray) and probably so in Western Australia (Atkinson). Cairns is the only place in Queensland from which a definite report was obtained (Moore). This is partly confirmed by the way the incidence decreases approaching the Queensland border.

We may, therefore, confine our attention to the south-eastern part of Australia, including Tasmania. In New South Wales and Victoria similar conditions are found; a series of mountain ranges and tablelands over 2,000 to 4,000 feet high extending from the coastal Macpherson Range at Point Danger (practically the Queensland border) to the Kilmore Gate due north of Melbourne in Victoria. We may say in general terms that thyreoid enlargement is seen in New South Wales in the coastal plains, especially that of the Hunter River Valley, on the valleys and slopes of the tableland to the east and close in the tableland to the west, all places with a considerable rainfall and many rainy days. This is also true of Victoria. Generally speaking, any considerable intensity is limited to the Gippsland plain and slopes and at Omeo immediately north of the main ranges, where heavy and frequent rainfalls are found.

Of Tasmania I speak without personal knowledge. It would appear, however, that similar conditions prevail, the difference being that it occurs in the west and south, King, Huon and Derwent Valleys, a few spots on the north edge of the western tiers (Deloraine), an incidence which corresponds to the heavier rainfall of west and south and steeper contours.

These ideas are confirmed by the fact that South Australia has in its settled portions no elevation

over 2,000 feet and no heavy rainfall and the same applies to Western Australia. Queensland, of which we know so little, has no part over 2,000 feet with steep contours except to the west and south-west of Brisbane and at Cairns.

These statements are emphasized by taking the localities where goître is known to be absent as much as by studying the goîtrous areas themselves. Association with occupations as dairy farming in goîtrous and sheep and cattle and wheat in non-goîtrous is probably a purely rainfall and not occupational phenomenon. Similarly that we find beeches and apples often grow in goîtrous areas.

All this appears in favour of the soil theory and the iodine or lack of iodine supply in diet. In all these areas people live to a large extent on the soil and so suffer according to its quality from this food deficiency. The idea that nearness to the ocean spells freedom may be due to the leaching action having not yet completely dealt with the iodides in the soil and is certainly not true of Bairnsdale in Victoria, a few miles only from the sea, or in Geeveston in Tasmania which is on a river estuary.

Efforts to obtain information regarding the incidence in animals, for example sheep, have so far been failures in this investigation.

The alternative theory is that of water carriage of infection. McCarrison's observations on springs and on fish are well known. Goître wells have been recorded by many observers. It is rather striking that within the areas of the Sydney and Newcastle water supplies goître is rare and the person with evident enlargement is usually imported. Of course, the fact that most of the food of such large cities is imported may meet this statement. We do, however, meet with people on the outskirts of towns, such as Bathurst, whose diet corresponds with those in the township itself, in whom thyreoid enlargement occurs, the only difference being that tank water was their supply and not the town filtered or reservoir supplies.

The second set of observations on location I submit with considerable diffidence. They are based on geological maps. The chief incidence appears confined to palæozoic strata: Permocarboniferous, Devonian, silurian and especially the former and the marine tertiary deposits derived from these rocks. This is particularly noticeable in the Hunter River Valley, the Gippsland area and the southern Tasmanian area. The marine tertiaries outlying from permocarboniferous areas with steep slopes and heavy rainfall form the most typical locations. Goître does not appear in association with trias-jura or cretaceous rocks to any extent in Australia. Magnesium, limestone and hard waters do not cause goître in Australia. Similar incidence has been noted in Europe. The explanation suggested concerning the association with rainfall is that low rainfall and few rainy days in the year produce soil conditions where the capillary action of percolated rain in the subsoil is greater than the leaching action.

During the actual fall of rain soluble iodides and other mineral salts are washed from the surface to deeper layers, but the evaporation on the much greater number of dry days keeps going a regular upward flow by capillarity of subsoil water with the soluble minerals in it to be deposited again in the surface subsoil by further evaporation. Heavy rainfall and many rainy days lead on the contrary to the leaching or washing away of soluble iodides first from hills and tablelands into the river valleys, then from the alluvial deposits themselves. Probably conditions with steeper contours and heavy rainfall will be still more prone to lead to this leaching process. This explains the fact in New South Wales and Victoria that the hot dry plains are very free from it and quite an abrupt line may be drawn on the western edges of the northern tablelands between goîtrous and non-goîtrous areas; for example Barraba and Manilla are goîtrous; Narrabri, fifty miles west, the area is non-goîtrous. On the other hand, tank water or surface water is used throughout large areas where thyreoid enlargement is unknown. The suggestion is that the *débris* from roof and spouting is sterilized on the roofs in these hot dry regions and not in the areas with constant heavy rains and so infection from the soil reaches the tank.

However, no one has yet isolated any definite medium of infection. It is difficult on soil and diet alone to explain individual cases in the actual areas following up one family after the other. As an example I quote some recent observations by Durie.

Goître is prevalent in Tamworth and Manilla, the soil is reddish, heavy with luxuriant growth; tank water is universally used for drinking purposes. Tamworth has a town water supply rarely if ever used for drinking. Gunnedah, thirty miles away, is flatter, the soil similar

Table I.—Goître in Western Australia, South Australia and Queensland.

(*From reports from the school medical officers.*)

State and District.	Medical Officer.	Number of Goîtres.	Number of Children.
Western Australia	Dr. R. H. M. Jull	11	14,584
South Australia	Dr. G. Halley	3	Large number, one year's total
Queensland—			
Gympie-Maryborough	Dr. E. Bourke	0	4,060
Country Districts	Dr. E. Sweet	0	7,457
Brisbane	Dr. E. Sweet	0	2,217
		1	1,966
Rockhampton	Dr. E. Sweet	1	950
Cairns	Dr. E. Sweet	0	520 boys
		22	609 girls

TABLE II.—GOÎTRE IN VICTORIA.

District.	Incidence.		
	Boys. (%)	Girls. (%)	Children. (%)
Bairnsdale—			
Primary School ..	33	35	—
High School	37·6	47·3	—
Sale	—	—	53
Warragul	—	—	36
Melbourne High School	—	—	13·2

Two severe cases out of thirty were reported from Warragul, three out of thirty from Sale and four out of twenty-two from Melbourne.

Goîtres were reported from Omeo, Warrnambool, Hamilton, Colac, Ballarat and Ararat. No goîtres were reported from Bendigo, Beechworth, Mansfield, Horsham, Mildura or Maryborough.

but sandier. The town water supply which is popular for drinking purposes, is very hard and mineralized and filters through sand from the river. The few cases of thyroid seen come from outside the town and drank tank water. Bingara with less incidence than Barraba uses deep well water, the latter tank water. At Narrabri, where thyroid is uncommon, a subartesian supply is used. Similarly in Moree where bore water and well water are mainly used. Inverell with some thyroid, tank water is used, the soil is partly red and heavy, partly black soil.

Artesian water supplies are not associated with goître in New South Wales.

In conclusion one or two observations may be made.

Firstly, the best test of a goîtrous area is its incidence in boys. Enlargement of the thyreoid in boys is always abnormal, it may be physiological in girls. The incidence in boys in proportion to girls rises rapidly as goître becomes more prevalent in an area.

Secondly, more evidence is desirable as to the association between enlargement of the thyreoid and exophthalmic goître. Exophthalmic goître often appears unrelated to simple enlargement and to goîtrous areas. Several observers (Devine, Ray, Cleland) have confirmed the observation that occurrence of exophthalmic goître does not follow the district incidence of thyreoid enlargement.

Among a number of teachers with enlargement of the thyreoid under observation during the past seven years, though a few had some hyperthyreoid symptoms and treatment was advised, none developed exophthalmic goître. Two teachers in whom exopthalmos developed, had not previously

TABLE III.—GOÎTRE IN TASMANIA.

(Incidence per 10,000 of population.)

District.	Incidence.	
	Males.	Females.
Derwent Valley—		
Both well	16	16
Oatlands	—	122
Hamilton	18	80
Green Ponds	—	34
New Norfolk	108	568
Brighton	—	—
Glenorchy	—	49
Hobart	2	25
Clarence	7	28
Huon Valley—		
Huon	34	232
Cygnet	14	45
Esperance	28	180
King River—		
Gormanston	10	133
Queenstown	12	422
Strahan	—	46
Other Districts—		
Brum	—	30
Deloraine	—	10
Devonport	—	9
Emu Bay	—	10
Fingal	—	12
Kentish	—	28
Kingborough	—	15
Latrobe	—	32
Lilydale	—	13
Longford	—	3
Leven	—	10
Scottsdale	—	42
Sorell	—	39
Spring Bay	—	24
Waratah	—	60

No goîtres were reported from Campbelltown, Circular Head, Evandale, Flinders, George Town, Glamorgan, King Island, Penguin, Portland, Richmond, Ringaroowa, Ross, Table Cape, Tasman, Westbury or Zeehan.

TABLE IV.—GOÎTRE IN AUSTRALIA.

Incidence per 10,000 of School Population.

School District.	Thyreoid Enlargement.		Severe Degree of Thyreoid Enlargement.	
	Boys.	Girls.	Boys.	Girls.
Tamworth	633	1,200	153	543
Muswellbrook	550	1,127	163	556
Grafton	42	435	14	51
Armidale	76	357	6	122
Maitland	57	301	11	66
Campbelltown	77	260	17	44
Newcastle North	23	228	14	51
Newcastle South	23	219	17	44
Yass	48	203	—	10
Albury	6	195	—	65
Casino	47	180	28	54
Braidwood	9	174	9	9
Lismore	18	159	—	4
Young	—	114	—	—
Bathurst	—	87	—	—
Taree	31	88	—	8
Kempsey	—	61	—	—
Goulburn	—	60	—	8
Bega	—	30	—	—
Inverell	—	28	—	20
Narrabri	—	17	—	8

had any noticeable enlargement nor did they come from goîtrous areas.

In school children enlargement of the gland is associated with good health; they are usually mentally bright and above standard weight for height. The incidence is well distributed throughout the population and no association with previous illnesses noted.

The chief objection is on the æsthetic side and certainly the swollen necks of many in goîtrous areas are objectionable. Perhaps the gland hypertrophy is as has been suggested one of physiological compensation though one does not feel that these enlargements can be treated lightly. The association with toxic thyreoid enlargement and exophthalmic goître has not yet been fully demonstrated.

Prevention on any considerable scale has not been tried properly in Australia. Some efforts are being made in Bairnsdale by the administration of iodine to school children. Iodized salt is already in existence in South Australia, but not in Victoria, Tasmania or New South Wales.

THE PATHOLOGY OF GOÎTRE.

By A. M. Drennan, M.D., F.R.C.P.E.,
Professor of Pathology, University of Otago, Dunedin, New Zealand.

In his paper preceding this and in a previous communication[1] my colleague Professor Hercus has shown very clearly that there is a definite relation in New Zealand between the distribution of goître and the deficiency of iodine in the soil and in products of the soil.

The relationship of iodine to goître has long been debated, but in spite of those who deny any relationship of cause and effect, I feel convinced that, so far as our present knowledge goes, iodine is the most important initial factor in the pathological condition we call goître. I shall endeavour to develope briefly this thesis in my subsequent remarks.

It is more than thirty years since Baumann[2] found iodine in human thyreoids and he noted a variation in amount in different cases. Since then many observers have investigated the iodine content of thyreoids, both human and animal.

Marine and Lenhart[3] first clearly showed a relationship between the histological structure of the thyreoid and its iodine content, while Rogoff and Marine[4] pointed out that the richer the gland was in iodine the richer it was in thyreoid hormone. From the result of much other work also it is safe to assume that the iodine of the thyreoid is a fair index of the hormone stored in it.

Some five years ago C. S. Hicks and I investigated the iodine content of a number of human thyreoids and compared the histological appearances. Hunter's method[5] was employed and, as the whole gland had to be used, after pieces for histology had been taken off, it was not possible to get an idea of the distribution of the iodine in different parts of the gland.

During the last two years K. C. Roberts has been using von Fellenberg's method[6] for iodine in foodstuffs and he found it possible to examine portions of thyreoid, so that one-half could be used for chemical analysis and the other half of the same kind of tissue used for histological examination.

In examining any number of goître specimens one is soon impressed by the difficulty Marine noted, namely, that in the fully developed goître there may be an infinite variety of pathological changes seen. It is necessary, therefore, to study the earlier changes where possible and to examine *post mortem* thyreoids where no question of goître has ever arisen clinically, if one is to attempt to unravel the changes of the fully developed goître.

In going over specimens of goîtres sent in to the laboratory I found the following histological changes, expressed in Marine's terms[7] (see Table I.).

Table I.

Histological Change.	Group I (Simple.)	Group II. (Toxic.)	Group III. (Exophthalmic.)
Atrophy	34 = 59·12%	30 = 51·72%	11 = 26·82%
Colloid	50 = 87·71%	49 = 84·48%	22 = 53·65%
Hyperplastic Colloid	25 = 43·85%	21 = 36·20%	22 = 53·65%
Hyperplasia	5 = 8·77%	21 = 36·20%	27 = 65·85%
Adenoma	9 = 15·78%	26 = 48·82%	2 = 4·87%
Degeneration	14 = 24·56%	18 = 31·63%	6 = 14·63%
Fibrosis	38 = 66·66%	40 = 68·96%	15 = 36·58%
Hæmorrhage	30 = 52·63%	29 = 50·00%	13 = 31·70%
Total number	57	58	41

These groups are based on the clinical diagnosis and in Group III (exophthalmic) occur both primary and secondary types of exophthalmic goître; still the findings indicate a higher proportion of colloid tissue in the "simple" type than in the "toxic," while "hyperplastic" ("secretory" of Scott Williamson and Pearse[8]) appearances increase progressively from the "simple" to the "exophthalmic" type.

When the iodine content of such glands was compared, Hicks and I found that there was great variation, but certain general facts emerged, namely, the more extensive the "hyperplastic" change the lower the iodine content per gramme of gland substance; further, in both "simple" and "toxic" types the total estimated iodine in the gland was frequently large even though the amount per gramme was low; this was due to the large mass of gland tissue in many such cases. There were, however, many discrepancies which we could not explain satisfactorily.

By means of von Fellenberg's method Roberts and I were enabled to make what might be described as a fractional analysis of the different parts of any gland and the results have proved very interesting.

In investigating the iodine content of any thyreoid it is necessary to know if iodine has been

FIGURE I.

given to the patient before the gland was removed; iodine so exhibited may definitely raise the iodine content of the gland substance, a fact also noted by Tobler.[9]

The following few examples will make the subsequent summing up easier to follow and indicate the nature of the data on which such summing up is based.

Diffuse Colloid "Toxic" Goître.

1. (2/2780).—A girl, aged seventeen years. Goître was noticed for one year; definite toxic symptoms were present. A small amount of iodine was given before operation. Approximately two-thirds of one lobe was removed.

The total weight of the fresh specimen was 44·0 grammes. The whole gland must, therefore, have been much enlarged.

Macroscopically the specimen had a uniform, coarsely granular surface composed of colloid lobules. No "adenomas," cysts or hæmorrhages were visible in the gland substance (see Figure I).

Microscopically it was seen to contain alveoli varying greatly in size from very small to large and irregular, but all with flat to low cubical epithelium and dense colloid content. In parts there was fibrosis of the stroma in which atrophied alveoli occurred.

Histologically it was a diffuse colloid type of goître (see Figure II).

The iodine content was 0·67 milligramme per gramme of fresh gland, or, expressed in microgrammes as used by Dr. Hercus in soil and food iodine contents, this becomes 670 γ per gramme of fresh gland, a high unit amount.

The total iodine in such a gland must be very large. If forty-four grammes be taken as two-thirds of one lobe it gives sixty-six grammes for that lobe; at a conservative estimate the other lobe would be not less than forty grammes, say at a minimum one hundred grammes for the whole gland. That would mean sixty-seven milligrammes or 67,000 microgrammes of iodine in the whole gland.

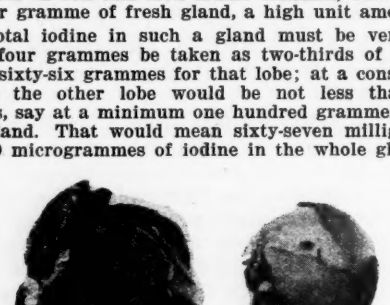


FIGURE II.

Nodular Hyperplastic "Toxic" Goître ("Toxic Adenoma" Type).

2. (3/408).—The patient was a married woman, aged forty-five. Goître had been noticed for ten years, causing pressure and dyspnœa, later mild toxic symptoms. No iodine was given before operation. Two pieces were removed. The total weight of fresh specimen was seventeen grammes. Of the two pieces one (*b*) was pale pink, soft and fleshy, with ill defined edges; the other (*a*) was red and fleshy, resembling normal gland (see Figure III).

The iodine content of (*a*) was 0·55 milligramme or 550 microgrammes per gramme of fresh tissue; of (*b*) it was 0·04 milligramme or 40 microgrammes per gramme of fresh tissue.

Microscopically (*a*) was found to consist of small colloid alveoli with flat epithelium; the septa were thickened and there was a slight, diffuse, interalveolar fibrosis; the blood vessels were engorged (see Figure IV). Specimen (*b*) was composed of larger colloid alveoli with flat epithelium and fibrous overgrowth in parts; these merged with large masses of closely packed alveoli as solid clumps or cords of cells ("fœtal adenoma" appearance); at one part there was atrophy of these cords and the stroma was fibrous and excessive (see Figure V).

This is a very common type of goître where parts are atrophied, parts in the resting, colloid state and parts actively hyperplastic or secretory in type, forming the so-called "fœtal adenoma" of the literature. The iodine contents of the two parts are in marked contrast. The total mass of this goître was apparently much less than many and the toxic symptoms were minimal.

Diffuse Hyperplastic "Exophthalmic" Goître.

3. (3/1368).—This patient was a man aged forty. Clinically he presented the typical symptoms of exophthalmic goître. There had been rapid loss of weight for the last six months. Basal metabolic rate was + 75. While in hospital he was given Lugol's solution (0·6 mil or ten minims twice daily) for fourteen days and there was definite clinical improvement; the pulse came down from 140 to 80. At operation most of both lobes was removed.

The total fresh weight of the specimen was 69·0 grammes; it consisted of the greater part of one lobe and about half of the other. The surface was smooth, the cut surface was uniform, pale pink and cloudy in appearance. All areas were similar (see Figure VI).

The iodine content was 0·21 milligramme per gramme of fresh gland or 210 microgrammes.

Microscopically the lobules were seen to be composed of small solid alveoli; in some lobules these were mixed with larger irregular alveoli with a thin content and high epithelium, in these alveoli occurred projecting tufts of epithelium. By the method of Williamson and Pearse the intracellular channels described by them were seen.

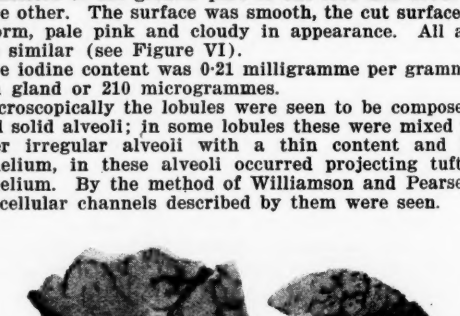


FIGURE VI.

FIGURE VIII.

There was no fibrosis and no congestion. Occasionally groups of lymphocytes were visible amongst the epithelium masses and in places the epithelium of some alveoli was shed off (see Figure VII).

This case is an example of the primary exophthalmic type of goître. Histologically it is an example of the diffuse hyperplastic goître or, in the terminology of Williamson and Pearse[10], it is an heterotrophic gland, an "adenoid goître," the epithelium being in a state of active secretion.

For a gland of this type the iodine content is relatively high, but iodine had been given in the form of Lugol's solution for a fortnight before the gland was removed. Again the size of the gland is to be noted, sixty-nine grammes were removed representing approximately three-quarters of the whole gland which would therefore weigh about ninety grammes. When the histological structure is remembered, it will be seen that there was a great excess of active secreting epithelium present.

Diffuse Hyperplastic "Exophthalmic" Goître.

4. (3/735).—This patient was a married woman, aged thirty-seven, who yielded a typical clinical picture of exophthalmic goître. Iodine was given over a period of six weeks with occasional intermissions before operation.

Two portions were removed, namely, the greater part of one lobe and a small part of the other lobe; the total weight of the two portions being 36·0 grammes.

Both portions were similar, macroscopically and microscopically. The cut surface was a uniform opaque pale pink (see Figure VIII).

Microscopically the lobules were found to be composed of small to medium sized alveoli with a thin granular or vacuolated content and high cubical to columnar epithelium. The capillaries were congested. There was no fibrous increase. There were scattered small collections of lymphocytes amongst the alveoli (see Figure IX).

The iodine content was 0·5 milligramme (500 microgrammes) per gramme of fresh gland in the larger specimen and 0·6 milligramme (600 microgrammes) in the smaller specimen.

The main features of this case are similar to the preceding one, No. 3, but iodine had been given in larger amounts and over a longer period; the iodine content of the tissue also was three times as high as in No. 3.

Further the histological appearances are similar to those described by Reinhoff[11] in cases of primary exophthalmic goître in which the gland was examined histologically before and after iodine administration. It is the picture of a gland where storage is being attempted, even though the epithelium is still active.

Diffuse Hyperplastic "Toxic" Goître.

5. (3/683).—This patient was a married woman, aged thirty-three, who had been aware of a goître for three years. She lost weight, had tremor and a rapid pulse but no exophthalmos. Her condition was regarded clinically as a "toxic" goître. No iodine was given.

The specimen of gland removed consisted of approximately half of each lobe with a portion of isthmus attached to one lobe. All portions were similar in character, being of uniform pale pinkish grey opaque gland tissue (see Figure X). The total weight of the fresh specimen was 34·0 grammes.

The iodine content was the same in each lobe, namely 0·06 milligramme (sixty microgrammes) per gramme of fresh gland.

Microscopically the alveoli were seen to vary considerably in size from large to very small. The alveoli contained granular or vacuolated content and were lined by high cubical to columnar epithelium. Mixed with the larger alveoli were many small, solid alveoli with large clear polygonal cells. The blood vessels were not engorged. There was no increase of interstitial tissue. There were many collections of lymphoid cells in the interstitial tissue and also amongst the epithelial cells, especially those in solid alveoli which were disintegrated at such places (see Figure XI).

The interest of this case is the low iodine content, the hyperplastic type of epithelium and the numerous collections of lymphocytes. It corres-

FIGURE X.

ponds to the "lymphadenoid" type of hyperplastic gland of Williamson and Pearse.[10]

These lymphoid collections I regard as evidence of exhaustion and commencing atrophy of the gland tissue; they occur in old degenerated goîtres, in some fatal exophthalmic cases and in analogous conditions elsewhere in the body.

Hyperplastic Colloid "Simple" Goître.

6. (3/1220).—This patient was a female, aged seventeen, who had noticed a swelling in the right side of the neck for one year and a swelling in the left side for two months. The swelling was rapidly increasing, the neck having increased two centimetres in two weeks. There was slight difficulty in swallowing, but no other clinical disturbance. The basal metabolic rate was normal.

Iodine had been given in the form of sodium iodide 0·03 gramme (half a grain) twice daily for five days before operation.

Parts of both lobes and isthmus were removed, the whole specimen weighing 275·0 grammes.

The three portions of the specimen all presented the same appearances, being smooth externally; the cut surface was uniform, watery, pale pink and granular from the large size of the lobules, an occasional clear, semi-fluid cyst was visible (see Figure XII).

The iodine content of different parts ranged from 0·24 milligramme (240 microgrammes) to 0·26 milligramme (260 microgrammes) per gramme of fresh gland.

Microscopically all parts appeared to be similar, containing the alveoli mostly very large and often irregular with a thin, uniform colloid content and cells ranging from low cubical to columnar, both types often occurring in the same alveolus at different places. In addition were groups amongst the above alveoli, of small hyperplastic alveoli with cubical irregular epithelium and no content or thin vacuolated material. The capillaries were congested but there was no increase of stroma (see Figure XIII).

This is a growing goître in a young patient who at the time of operation manifested no "toxic" symptoms. The mass of thyreoid tissue is very large and the iodine content is higher than would be expected from the histology alone and has been raised by the iodine administered.

By way of contrast is the next case No. 7.

Nodular Hyperplastic "Simple" Goître with Degenerative Changes ("Adenomatous" Goître).

7. 3/1479.—This patient was a married woman, aged forty-seven, who had had a goître for many years. Clinically the only disturbance was tracheal pressure causing some dyspnœa and difficulty in swallowing. The patient

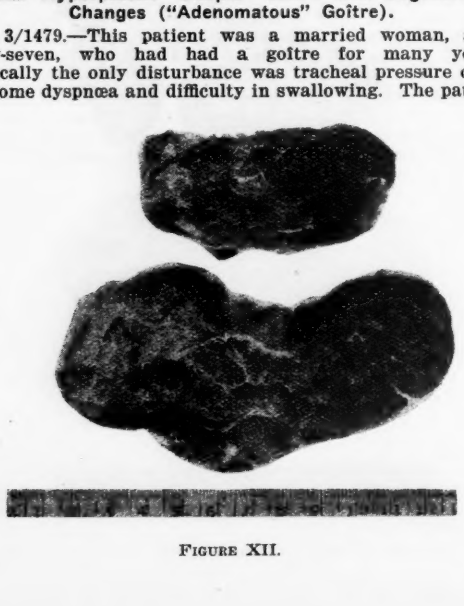


FIGURE XII.

FIGURE XIV.

came from a mildly endemic area. No iodine was given. About half of each lobe was removed surgically and the total specimen weighed 160 grammes.

The gross appearances varied; there was a main groundwork of coarse lobules, pinkish grey in colour with occasional small clear cysts. Amongst this mainly in the lower parts of both lobes were large, gelatinous, colloid nodules (adenomata) with degenerative changes in their centres (see Figure XIV). The following parts were examined histologically and chemically: (*a*) A clear nodule in part of a coarse lobule, (*b*), (*c*) part of a coarse opaque lobule, (*d*) a large gelatinous colloid nodule and (*e*) part of the coarse lobular groundwork from the other lobe. The iodine contents of these were: (*a*) 0·02 milligramme (20 microgrammes), (*b*) 0·02 milligramme (20 microgrammes), (*c*), (*d*) and (*e*) traces only.

Microscopically it was seen that (*a*) was composed of small alveoli of high epithelium, some of these being unusually large and no content or a thin vacuolated material frequently with shed off cells in it. The stroma was increased in parts, especially at the margins of groups of the above alveoli and in it were atrophied remains of alveoli and collections of lymphocytes. The vessels were congested (see Figure XV).

In (*b*) was similar tissue and also groups of large alveoli with cubical epithelium and colloid content. The appearances in (*c*) were very similar to (*a*) and (*b*) (see Figure XVI).

Specimen (*c*) consisted of lobules of small hyperplastic to hyperplastic colloid alveoli, similar to parts of (*a*); hæmorrhage was present in parts (see Figure XVII).

In (*d*) there were very large alveoli distended with thin colloid and lined by low epithelium; mixed with these and also forming groups by themselves were small alveoli mostly of hyperplastic type though an occasional colloid one also occurred. There were also large patches of scar tissue in which appeared remains of atrophied alveoli (see Figure XVIII).

In this example the low iodine is to be noted associated with mixed hyperplastic and colloid tissue, degenerated in many places. It is a gland that has responded to some stimulus and is now showing signs of atrophy and exhaustion.

Many more examples from patients operated on could be given, but space does not permit of them.

If *post mortem* material be examined, exactly the same types of gross and microscopical changes are found, even from patients in whom there have been no indications of goître or thyreoid disturbance whatever. Even the iodine distribution in the glands is similar to that found in definite clinical goître and varies according to iodine given. There is this

great difference, the glands dealt with *post mortem* have not the bulk of tissue reached by the clinical goîtres. This "mass effect" I regard as most important; it will be referred to again below.

A word about animal thyreoids. This aspect of the goître work is still incomplete here, so need be referred to only briefly as yet.

Roberts has found considerable normal variation in iodine content of animal thyreoids taken at the same period of the year, for example sheep thyreoids ranged from 0·28 milligramme (280 microgrammes) to 2·9 milligrammes (2,900 microgrammes) per gramme of fresh gland; the thyreoids of pigs varied from 0·3 milligramme to 0·6 milligramme (300 to 600 microgrammes) per gramme of fresh gland. Histologically these all reveal a very uniform colloid type of gland. Rats and rabbits seem to have normally a lower plane of iodine content. A wild rat gave an iodine content of 0·36 milligramme per gramme (360 microgrammes), a tame white rat on a normal laboratory diet 0·5 milligramme per gramme (500 microgrammes) and one on a high iodine diet showed 1·6 milligramme per gramme (1,600 microgrammes). A white rat of 260 grammes total weight on a low iodine diet, namely twenty microgrammes iodine per kilogram, had a large gland (thirty milligrammes against the normal ranging from nine to twenty milligrammes) with only 0·03 milligramme per gramme (30 microgrammes) of iodine. Histologically this gland (3/1631) (see Figures XIX and XX) is of the diffuse hyperplastic ("secretory") type with much shedding of epithelium and only one or two large dilated colloid alveoli; it is congested. Rats on the normal diets are of more colloid type (3/1760) (see Figure XXI).

One interesting point emerges in the sheep thyreoids, namely that the highest in iodine content was a thyreoid from a Taranaki (North Island) sheep, from an area of high soil iodine. Histologically the gland was of uniform colloid type similar to those with much lower iodine content.

The converse is seen in the case of a thyreoid from a wild hare from Omakau district of Central Otago, a low iodine area. This gland gave only 0·054 milligramme (54 microgrammes) per gramme of fresh gland. Histologically there was a general increase of fibrous tissue in this gland and very small colloid alveoli, an atrophic, fibrosed gland.

Can we sort out and rearrange this confusing mass of data and make a connected story from it?

I do not agree with Williamson and Pearse[10] that the clinical condition of the patient should be ignored and only the histological appearances used to group goîtres, except from a purely anatomical point of view. Nor can I see any particular advantage in the many new terms they employ in their pathological classification; it is true the term "hyperplasia" is not altogether satisfactory; their term of "secretory" is perhaps more suitable.

Personally I would recognize but two main phases: The resting, storing or colloid and the active, secretory or hyperplastic. Also, I feel that the evidence shows that one may merge into the other in spite of the contrary opinion of Williamson and Pearse. What these workers have done is to give a very fine description of the histology of the thyreoid, normal and pathological, but they have not attempted to explain the origin of such pathological appearances as they figure. They dismiss the idea of iodine deficiency as a basal cause of thyreoid disturbance without having studied the iodine factor. They conclude in the same unsatisfactory way as W. M. Ash[12] and others by saying that the cause of goître is still unknown.

Possibly it is, but I feel that the work that has been done in this school on the iodine factor, is an honest attempt to find a primary cause and an attempt that has been rewarded by some small measure of success.

Iodine deficiency may not be the only cause; there may be accessory factors modifying the effects of such deficiency, but that it is a very important primary cause of goître I feel certain from the results of the work here and elsewhere. Further I would suggest that this deficiency of iodine is the basic cause of all forms of goître, not merely the endemic but also the "toxic" and the "primary exophthalmic."

The sequence of events may be set out graphically in the following schema (see schema of goître).

The third column is one to which exception may be taken by many. It may be asked if iodine deficiency is a cause of this form, why is there not more of it in endemic areas? I can only say that there is much of this form here in New Zealand as indicated by the statistics given by Dr. Hercus, though in these there is no adequate distinction made between the primary exophthalmic goître and the secondary form complicating nodular or "adenomatous" forms. I have been struck by the number of primary exophthalmic goître thyreoids that we have examined in the ordinary routine without any selection of cases.

The main trouble is that we often do not know the antecedent conditions as regards iodine intake in these primary exophthalmic goître patients nor do we know the histological state of the gland before clinical symptoms commence.

I am satisfied that there is a "hyperplastic" or "active secretory" type, of gland without any clinical symptoms or signs of goître, for I have seen such glands *post mortem* from persons in whom there was no suggestion of goître before death. I have two such specimens from persons who died suddenly under anæsthesia and in whom there was lymphatism. In such glands the iodine content is relatively low.

With regard to the other two columns little need be said. They are probably only variants of the same type of response, general in the first column, patchy in the second.

The sequence of events is somewhat as follows:

The thyreoid is exposed to an iodine deficiency, either diminished supply or minimal supply and increased demand. If it is to maintain the neces-

sary supply of hormone, it must respond in one of two main ways: (i) It may uniformly increase the storage area to lay up a larger supply of colloid which is at a lower iodine concentration. To do this the alveoli increase in size by means of the cells increasing, if necessary, in number and filling the alveoli with colloid. The result is a large colloid gland in the main though parts may show active, "hyperplastic," or "secretory" cell groups. This gives the simple colloid goître and is a common response in the endemic form. It may, however, never attract attention clinically and may indeed remain quiescent for an indefinite period. It is a compensated gland, balanced towards its intake of iodine.

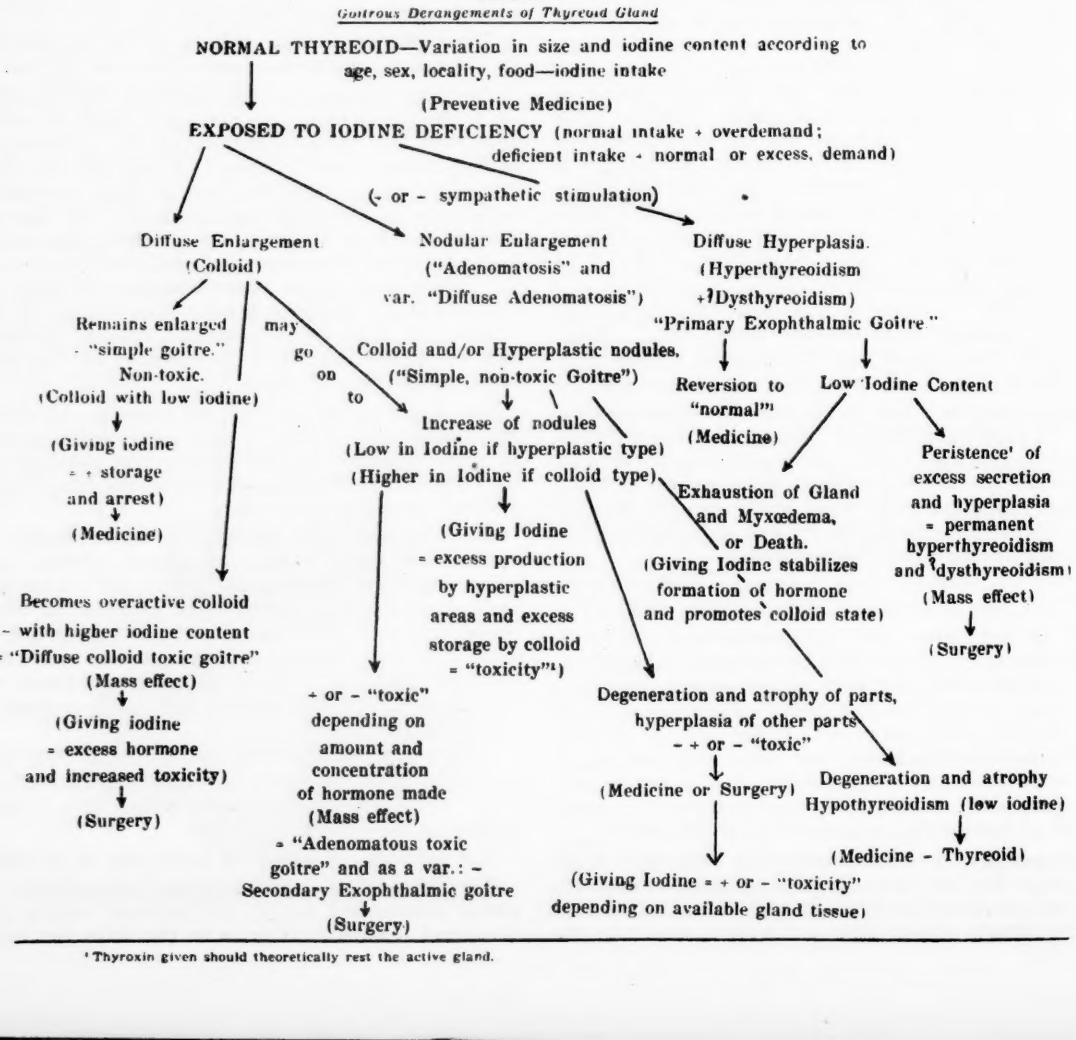
If the deficiency is sufficiently evident the gland may continue to enlarge and become noticeable, then all sorts of secondary effects are apt to occur; parts become exhausted and atrophy or hæmorrhages occur with subsequent organization, fibrosis and scar; other parts then hypertrophy or become "active secretory" and so the nodular or "adenomatous" form arises.

Such a large colloid gland has adapted itself to a low iodine intake and if excess of iodine be exhibited to this gland, it becomes overcharged with hormone and toxic symptoms are liable to appear; it becomes a diffuse colloid toxic goître. It is an example of mass effect. Fortunately "excess" is a relative term for, as shown by Hotz,[13] 700 microgrammes per day may be given to goîtrous persons without production of toxicity. The position might be likened to that where a twelve volt battery is used in a six volt lighting system of a motor car—the lamps cannot stand the strain, they burn out; even so with the body tissues when this large mass of fully charged thyreoid gland begins to discharge.

(ii) The thyreoid may respond to the initial deficiency by parts hypertrophying and laying up larger colloid stores or by parts becoming "active secretory" ("hyperplastic") and pouring out the hormone as it is made. So may arise the various forms of nodular or "adenomatous" goître. As indicated under (i) this nodular response may be a later change in an initial diffuse response by the exhaustion of parts and the activity of less damaged

Schema.

Goitrous Derangements of Thyreoid Gland



[1] Thyroxin given should theoretically rest the active gland.

parts; indeed many specimens suggest that this is the more likely origin of the nodular form, which thus becomes merely a variant of (i). Once such nodules appear, the secondary changes are legion, hæmorrhages, scarring, calcification and so forth and the toxicity or non-toxicity (or the over compensation or balance respectively) that result, depend on the mass of active secreting tissue or the concentration of iodine-containing hormone in the colloid nodules.

Iodine exhibited to such a gland, if the mass effect is sufficient, may produce definite "toxicity." The active secretory areas ("toxic adenomata") simply make up the iodine into hormone and pass it on into circulation; the resting colloid parts become overcharged and there occurs again the diffuse colloid effect mentioned before, but on a smaller scale, as regards the colloid part of such gland.

(iii) The other main mode of response to the iodine deficiency is for the whole gland to become active secretory, a diffuse "hyperplastic" gland.

This may compensate, but it gives no reserve store; any extra demand for hormone or need for more hormone, for example increased physical or psychical activity, or further diminution of iodine supply, means that the gland must increase its area or its activity or both; it becomes a larger active gland and somewhere at this point it may become of clinical interest as a "primary exophthalmic goître." The further progress of such a gland varies. If the demand is reduced, in time the gland may abate its activity and symptoms may disappear. If the gland has gone too far on its way, it may exhaust both itself and its owner and death may result or short of that, myxœdema may appear.

Iodine presented to such a gland appears to steady its activity and lead to some attempt at storage. There are no colloid areas here as in the nodular form and such storage takes place in the "hyperplastic" or "secretory" parts. This is shown in our series and is well brought out in Reinhoff's cases.

But clinical experience shows that while iodine may improve the clinical condition and reduce the basal metabolic rate, it will not effect a permanent cure. This is what one would expect from the pathological appearances. The overactivity is partly arrested by the iodine and some degree of storage established, but the gland is too large and the mass effect is again seen in that this enlarged and now semi-active gland has an over-rich hormone load which, as it is gradually unloaded, once more produces toxic effects.

In the schema I have ventured to put suggestions for treatment of the various forms of goître, based on this conception of the pathology.

It is futile to present iodine to a great mass of thyreoid tissue, especially as it is impossible clinically to know the character of that tissue. Iodine may be actually harmful in such cases as indicated above. The place of iodine is in prevention as Dr. Hercus has pointed out and the earlier the better.

It would be more rational to exhibit thyreoid extract itself or thyroxin in many goître patients, the difficulty would be to determine the exact amount required. Loeb[14] has shown experimentally in guinea pigs that iodine does not inhibit hypertrophy after partial resection of the gland, but thyroxin does inhibit such hypertrophy.

Plummer[15] has shown how the tissues use up a daily amount of thyroxin averaging from 0·5 to 1·0 milligramme and how "a shift of one milligramme of thyroxin in the tissues of the body is accompanied by a corresponding rise or fall of between 2% and 3% in the basal metabolism."

Thyroxin administered would appear to spare the thyreoid the necessity of making it form the raw materials and the gland may thus be rested. Iodine administered requires the exertion on the part of the thyreoid to prepare hormone from it.

It may be that by further development of basal metabolic rate examinations thyroxin will be of use in a certain number of goîtres. But, until prevention has eliminated goître, the surgeon will still have the lion's share in removing the masses of pathological thyreoid tissue that disfigure the necks and destroy the bodily mechanism of so many people today.

References.

(1) C. E. Hercus, W. N. Benson and C. L. Carter: "Endemic Goître in New Zealand and its Relations to the Soil Iodine," *The Journal of Hygiene*, December 17, 1925, page 391.

(2) Baumann: *Zeitschrift für Physiologische Chemie, Band* XXI, 1895-1896, *Seite* 319; *Ibidem, Band* XXII, 1896-1897, *Seite* 1.

(3) D. Marine and C. H. Lenhart: "Relation of Iodine to the Structure of Human Thyroids," *Archives of Internal Medicine*, November, 1909, Volume IV, page 440.

(4) J. M. Rogoff and D. Marine: "Attempts to Produce a Substance with Thyroid-like Activity by the Artificial Iodization of Proteins," *Journal of Pharmacology and Experimental Therapeutics*, Volume X, 1917-1918, page 321.

(5) A. Hunter: "The Determination of Small Quantities of Iodine, with Special Reference to the Iodine Content of the Thyroid Gland," *Journal of Biological Chemistry*, Volume VII, Number 5, May, 1910.

(6) T. von Fellenberg: "*Untersuchungen von jodierten Salzen*," *Biochemische Zeitschrift, Band* CXLII, 1923, *Seite* 263.

(7) D. Marine and C. H. Lenhart: "The Pathological Anatomy of the Human Thyroid Gland," *Archives of Internal Medicine*, April, 1911, Volume VII, page 506.

(8) G. S. Williamson and I. H. Pearse: "The Structure of the Thyroid Organ in Man," *Journal of Pathology and Bacteriology*, 1925, Volume XXVI, page 459.

(9) T. Tobler: "*Chemische und Histologische Untersuchungen am Strumen mit Besonderer Berücksichtigung von Jodbasedowfällen*," *Mitteillungen aus dem Grenzgebiet der Medizin und Chirurgie, Band* XXXVII, 1923-1924, *Seite* 622 (abstract in *The Journal of the American Medical Association*, 1924, page 724).

(10) G. S. Williamson and I. H. Pearse: "The Pathological Classification of Goître," *Journal of Pathology and Bacteriology*, Volume XXVIII, 1925, page 361.

(11) W. F. Reinhoff: "The Histological Changes Brought About in Cases of Exophthalmic Goître by the Administration of Iodine," *Johns Hopkins Hospital Bulletin*, Volume XXXVII, 1925, page 258.

(12) W. M. Ash: "Goître," *Journal of State Medicine*, November, 1926, page 637.

(13) G. Hotz (quoted by Hercus, Benson and Carter, *loco citato*).

(14) L. Loeb: "Studies on Compensatory Hypertrophy of the Thyroid Gland: IV," *Journal of Medical Research*, 1919-1920, Volume XLI, page 481; *ibidem* V, 1920-1921, Volume XLII, page 77.

(15) H. S. Plummer: "Interrelationship of Function of the Thyroid Gland and of its Active Agent, Thyroxin, in the Tissues of the Body," *Collected Papers of the Mayo Clinic*, Volume XIII, 1921, page 457.

THE MEDICAL TREATMENT OF GOITRE.

By R. R. STAWELL, M.D. (Melbourne), D.P.H. (London),
Honorary Consulting Physician, Melbourne Hospital; Honorary Consulting Physician, Children's Hospital, Melbourne.

IN considering and debating the medical treatment of goître, we must be clear as to what we mean by goître in its different forms; I shall adopt for this purpose the clinical classification suggested by Plummer.

CLASSIFICATION.

1. Simple or diffuse colloid or endemic goître.
2. Adenoma in a simple goître.
3. Adenoma in a simple goître with "hyperthyreoidism."
4. Exophthalmic goître.

Those who have read Plummer's recent Beaumont lecture will feel, with me, how clearly and convincingly he has expounded the reasonableness of this classification and all of us must have recognized for some years the helpfulness gained in our clinical work by the adoption of this classification. Those who have also studied the wonderful work of Williamson and Pearse, need not let themselves be confused by the introduction of new views on the morbid anatomy of the thyreoid gland or by new terms in the classification of goître.

It is well to realize that clinical classification, based alone on the morbid anatomy of the affected organ, may be more misleading than helpful in clinical work, as witness our experience in the past in dealing with disease of the kidney.

Simple Goître.

Uniform enlargement of the thyreoid gland, without any definite constitutional disturbance, occurring endemically or seen sporadically in young people is classed as "simple," diffuse colloid goître.

Since Marine's work of more than six years ago it has been generally recognized that the dominant factor in the causation of this condition, as we see it here, is an inadequate supply of iodine to the thyreoid gland. There must, of course, be other factors which I need not discuss now, but I must discuss certain points in the diagnosis of the condition.

Diagnosis of Simple Goître.

Though simple goître occurs without definite constitutional disturbances, there may be, on the one hand, symptoms of defective thyroxin output, such as listlessness and a dry skin, the direct result of the goître; there may be, on the other hand, "nervous" symptoms, such as tachycardia, sweating and occasional tremor, the indirect result of the goître.

If either of these two opposite conditions is found, the diagnosis and the treatment rest in these days on the findings of the basal metabolic rate.

Plummer states that in a large percentage of these cases, suggestive of hypothyreoidism, the basal metabolism is below the average normal; even if the symptoms are those of irritability and instability of the sympathetic nervous system, the basal metabolism may be normal or even subnormal; it may be raised, but if raised, it rapidly subsides under management. Such nervous symptoms cannot be regarded as directly associated with abnormal thyreoid functions. They are due to other factors, for instance distress and conflict of mind incidental to any indefinite and disfiguring form of ill-health in any sensitive adolescent.

If the basal metabolism rate remains high in what appears to be a simple goître, there is present a degree of active adenomatous tissue mixed up with the diffuse colloid goître.

Treatment of Simple Goître.

The essential plan of treatment of simple goître in these days and in these parts seems to me to consist in the administration of iodine or thyreoid gland extract or both and in severe cases the occasional use of thyroxin given intravenously.

Broadly speaking, if the metabolic rate is within normal limits, iodine in some form may be given with the hope of some improvement, though in cases with well established simple goître the degree of the improvement by the use of iodine alone has been in my experience disappointing; yet it is still recommended by such authorities as Dunhill and Fraser in their recent review of the treatment of goître. Iodine may be found to prevent simple goître, but I have been disappointed in it as a cure. If the metabolic rate is raised, it is advisable to avoid the ordinary iodine preparations, for they may irritate or activate an already smouldering adenoma. Desiccated thyreoid should then be given, with the idea that it supplements but does not stimulate the action of the gland. It is obvious that if the basal metabolism is below normal, it is imperatively necessary to give thyreoid gland extract. The dose must be determined in the first instance by the determination of the basal metabolic rate.

Plummer recently summarized his experience in the treatment of simple goître as follows:

> Desiccated thyroid is given in sufficient quantity to hold the metabolic rate at or a little above normal. At intervals of from three to six months the desiccated thyroid is stopped and five to ten drops of Lugol's solution used daily. If the thyroid again becomes distended with colloid, administration of the desiccated thyroid is resumed for another period; if not, the Lugol's solution is continued for a few months.

In my experience the treatment with thyreoid gland extract has been satisfactory and I know of a most dramatic improvement in a case in which an enlarging pulsating diffuse colloid goître, causing urgent symptoms of pressure dyspnœa shrank to about half its size after one injection of five milligrammes of thyroxin given intravenously.

It is, I think, wise to recognize the fact that the medical treatment of a simple goître is full of complexities. Not only is iodine frequently ineffective, but if there is a commencing adenoma, it may be harmful. The treatment with desiccated thyreoid requires careful observation and control and if there is a commencing "dysthyreoidism" associated with the simple goître, then treatment by thyreoid extract is harmful, while iodine may be of temporary benefit.

Following the suggestions of the workers in the Mayo Clinic, both in the treatment of simple goître and more recently in exophthalmic goître, I have commonly employed Lugol's solution of iodine. This solution is 5% iodine in a 10% aqueous solution of potassium iodide. In London, following Professor Fraser's suggestion, a 10% tincture of iodine made with 95% alcohol and without any potassium iodide is used. It is clear that such a solution would deposit the iodine as soon as it was mixed with milk or water, but this apparently has no appreciable disadvantage, for just as striking results have been obtained by one solution as the other. I have noticed that the Fraser's "French tincture of iodine" is less liable to produce an iodide rash than Lugol's solution. The dose of iodine must be allowed to vary enormously, according to the condition of the patient, as I shall explain later.

Adenoma in Simple Goître.

Before passing on to the consideration of the medical treatment of exophthalmic goître, I may state that there is no medical treatment for adenoma of the goître; it is entirely a surgical problem and as regards the treatment of adenoma of the thyreoid with "hyperthyreoidism," almost the same may be said.

Adenoma with Hyperthyreoidism.

In adenoma with hyperthyreoidism which for twenty years or so has been recognized as "toxic adenoma," the incidence of the toxæmia seems to affect the heart especially, with the oncoming of auricular fibrillation and heart failure. Twenty years ago, I "prepared" such patients for operation by Dr. Dunhill; the preparation consisted then in rest and full doses of digitalis and I thought such preparation was of help. The surgical results were certainly wonderfully good, considering the grave condition of the patient. Now, I must quote Plummer's summary on this particular subject:

> The treatment of hyperfunctioning adenomatous thyroid is essentially surgical. The operative mortality for a long series of years has been approximately 3·5%. The mortality for 1925 was approximately 1%. This drop in mortality was largely, if not wholly, due to stopping the use of digitalis in cases in which there was auricular fibrillation.

I do not fully understand that last sentence. It probably means that valuable time is lost by delaying operation, until a course of digitalis has been given. While on this subject, I may say that in both toxic adenoma and exophthalmic goître, I have seen no benefit, but some harm from the use of quinidine in the treatment of auricular fibrillation before the surgical removal of the source of the toxic substance; though cases have been recorded, indicating the contrary view.

Exophthalmic Goître.

Symptoms and Diagnosis.

As we all know, we are indebted to Plummer for many illuminating suggestions and hypotheses concerning the pathology of exophthalmic goître, but in no way are we more indebted to him than for his discriminating clinical observations as to the essentially characteristic symptoms of that disease. Now these special symptoms are not tachycardia nor sweating nor loss of weight nor other symptoms and signs of a raised metabolic rate, for such symptoms are also common in the "hyperthyreoid state" of a toxic adenoma. The essentially characteristic symptoms of exophthalmic goître are the combination of (i.) ocular signs, (ii.) a special syndrome of nervous symptoms and (iii.) the occurrence of crises during the natural course of the disease, when all these abnormal symptoms are exaggerated in intensity.

Plummer attributes this "complex" to a hypothetical abnormal iodine-deficient product of the thyreoid, producing these "dysthyreoidal" symptoms and associated with dysthyreoidal symptoms are the ordinary symptoms of hyperthyreoidism.

Ocular Signs.—The ocular signs are not only the exophthalmos which distinguishes by name this form of goître, but also a peculiar stare which is present with or without exophthalmos. Patients are often subjectively conscious of this peculiar stare, before an observer can recognize it as Stellwag's sign.

Nervous Phenomena.—The toxæmia makes the patient restless in a peculiar way, suddenly and irregularly hyperirritable and emotional. The restlessness takes the form "of purposeful but useless movements," such as buttoning or unbuttoning a garment, turning from side to side when sitting, constant changing from the recumbent to the partially sitting posture when under examination on a couch. With all this restlessness, there is a real weakness of muscular movements. This loss of strength is one of the earliest symptoms and is noticed on going upstairs or can be tested by stepping upon a chair. The weakness of the quadriceps muscle groups is so specific and characteristic of the condition, that it has diagnostic significance.

Crises.—The occurrence of crises of a profound toxæmia during the course of ill-health is the terrible feature of Graves's disease. The physical and mental restlessness may reach the intensity known as maniacal. The symptoms of sudden toxæmia range from attacks of nausea, vomiting and diarrhœa, up to the condition of delirium, coma and death.

Treatment.

Expectant Method.—As regards the medical treatment of exophthalmic goître, I would say from my own long experience that up to a little more than twenty years ago, treatment was ineffective. Sedatives and rest were ordered and the medical management of these cases followed the "expectant method." Certainly then, as now, in some cases the symptoms subsided after a long and disabling period of ill-health, but in this, the first period of my experience, I realized the ineffectiveness rather than the helpfulness of the methods adopted.

Introduction of Surgical Measures.—In 1905 Dr. T. P. Dunhill, then of Melbourne and now world-renowned for his thyreoid surgery, introduced the operation of partial thyreoidectomy under local anæsthesia for the more severe cases of Graves's disease. His results were so strikingly good in so

many individual patients, as I actually saw them, that my rôle as a physician was changed from indefinitely "waiting and seeing" to limiting definitely the duration of rest and sedative treatment, even in favourable cases to a period not longer than a year. In severe cases operation was advised without unreasonable delay. This plan of treatment constituted the second period of my experience.

Removal of Focal Infection.—Then, as now, search was being made for the cause of Graves's disease and the suggestion that bacterial toxæmia might be a factor in the causation began to be generally accepted some twelve years ago. I think I am right in saying that in certain milder forms of the disease, the surgical removal of infected tonsils or similar foci has led to or has been followed by improvement and as far as I know to a permanent subsidence of dysthyreoidal symptoms.

My present opinion is that in early Graves's disease, with or without appreciable enlargement of the thyreoid, such procedure is of value, but in well marked cases, it is, taken alone, useless. Indeed, I am inclined to the opinion that during a *crisis status* in goître there is such a weakening of resistance that acute or subacute general or local infections are particularly prone to occur and that to remove, for instance, inflamed tonsils at such a period would be most unwise and to remove chronic dental infections at or about that time would be most unreasonable. First lessen or lower the dysthyreoidism, then deal with extraneous burdens of ill-health that may be surgically removable.

X Ray Treatment.—In those days, as in these, the surgical mortality in very serious forms of exophthalmic goître led to the adoption of X ray treatment for this condition. My own experience and observations have led me to believe that in the milder, more favourable but yet progressing forms of this disease, skilled surgery is free from any appreciable risk, that in just these cases, X ray treatment is a time-consuming and time-wasting process, doubtful as to effectiveness and occasionally followed by serious symptoms of diminished function of the normal thyreoid tissue. In the cases that are or used to be a real "surgical risk," I have not seen X ray treatment produce any good result, but the delay associated with its ineffective use has raised the "surgical risk," which had to be taken later.

Food and Feeding.—During the latter part of my second period of experience, with the routine use of the knowledge of the caloric value and constituents of ordinary foods necessary in treating diabetes and with the coming into use of the metabolic rate estimations, I followed Boothby's plan of accurately, instead of vaguely, "feeding up" these patients, giving approximately two grammes of protein per kilogram of body weight and making up the rest of the caloric needs with a high proportion of fat and the necessary amount of carbohydrate.

Recently Professor Mellanby seems to suggest that from experimental work on animals it is disadvantageous to maintain this high caloric value in the food. In this matter, the proof of the pudding, no matter how compounded, is in the gain in weight of the patient.

"Modern" Treatment.—As regards the treatment of exophthalmic goître during the last two years a third period of my experience commenced in the use of some form of iodine in that disease. Though there were other workers in the same field, this method of treatment may be said to have been introduced by Plummer as the result of work and study at the Mayo clinic. The chemical theory which justified the introduction of iodine as part of the treatment of a disease supposed up to that time to be a manifestation of hyperthyreoidism, I cannot discuss now; but of the value of iodine as an agent in diminishing for a time at least certain of the special toxic effects of exophthalmic goître, I have no doubt. This action is so marked that it is known as the "iodine remission." The routine treatment is 0·6 mil (ten minims) of Lugol's solution three times daily for ten days and from that time on 0·6 mil is given only once a day, except during a crisis in the course of the disease, when six mils (one hundred minims) are given in divided doses by mouth or rectum within one or two hours. It must be recognized that the amount of iodine has to be carefully adapted to the needs of each patient, that in severe cases of the disease it puts the patient into a better state to stand the operation of subtotal thyreoidectomy which is, so far as my experience goes, the essential plan of treatment in definitely developed and persisting degrees of exophthalmic goître.

Formerly, my attitude to these cases was to "wait and see" and if no improvement occurred within a reasonable time, I advised operation. Now my rôle as a physician is to prepare these patients for the surgeon and to reinforce the effects of subtotal thyreoidectomy. In this preparation period we must aim at the control of the special nervous symptoms and concurrently obtain a lowered metabolic rate.

During the preparation period the pulse rate may be taken as indicative of the variation in metabolic rate.

During this time of observation the larynx should be specially examined to see that there is no evidence of impairment of either of the recurrent laryngeal nerves, for this condition has proved to be a grave contraindication to the operation as ordinarily performed and last of all I would venture to place before you an important contribution towards our knowledge of treatment of this disease. At a recent medical meeting of the staff of the Melbourne Hospital, Mr. Alan Newton showed on the basis of the hospital records that of all methods of general anæsthesia at present in use, the gas and oxygen is greatly to be preferred for these patients.

In conclusion, I believe that until we know the real causes of the graver forms of goître, it is the duty of the physician to recognize the limitations of medicine and to advise operation frankly as the essential plan of treatment.

Wholly Set Up, Published and Printed at The Printing House, Seamer Street, Glebe, Sydney, N.S.W., by HENRY WILLIAM ARMIT, of The Point Road, Woolwich, Sydney, on behalf of THE AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED, of 30-34, Elizabeth Street, Sydney.